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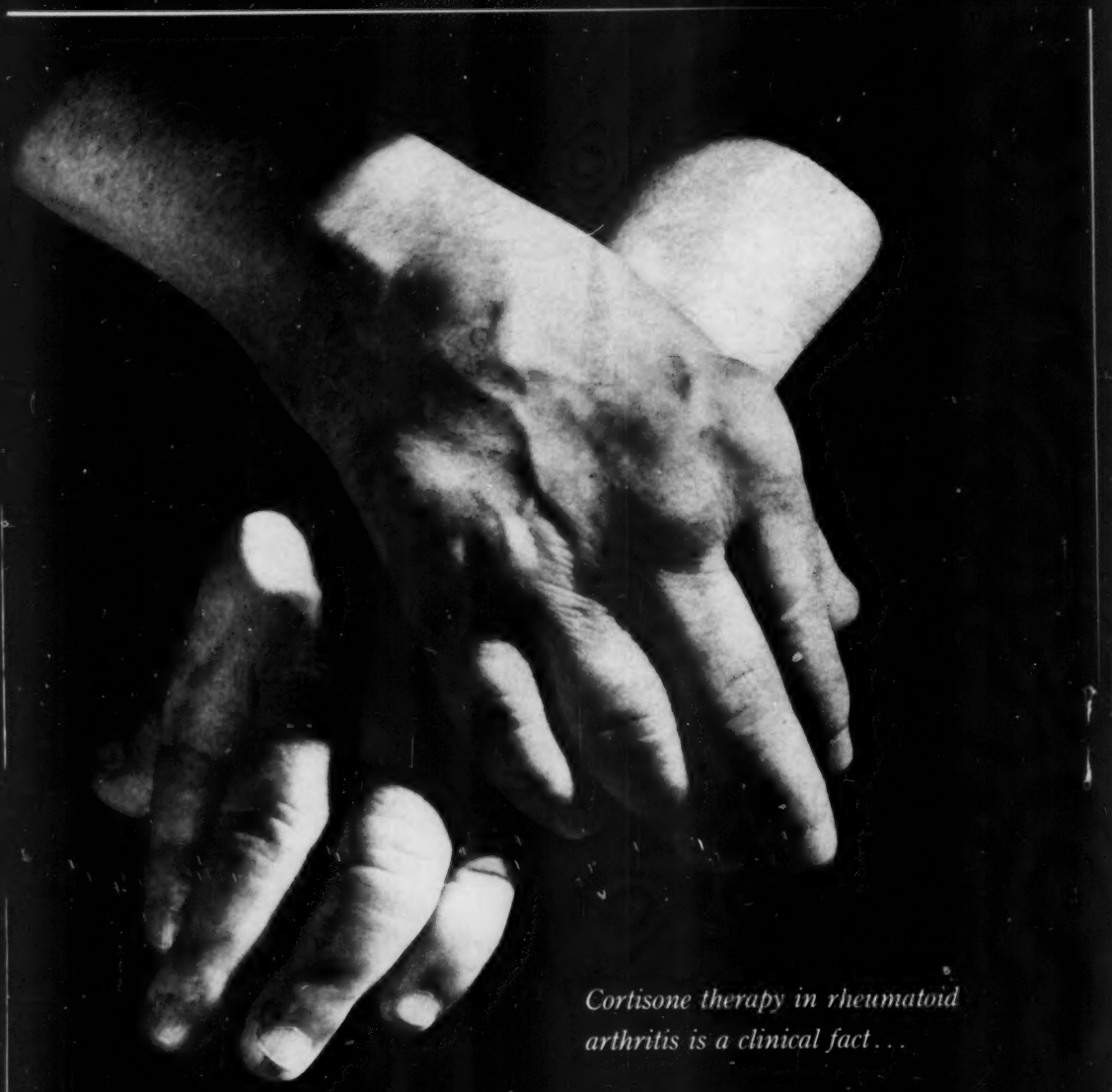

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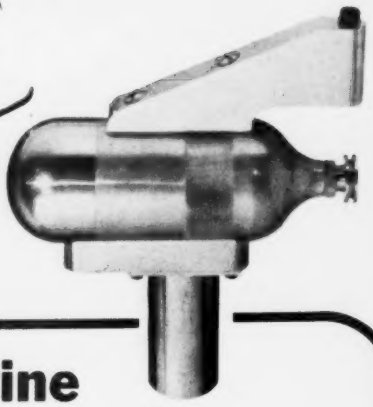

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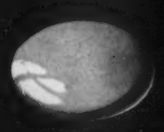

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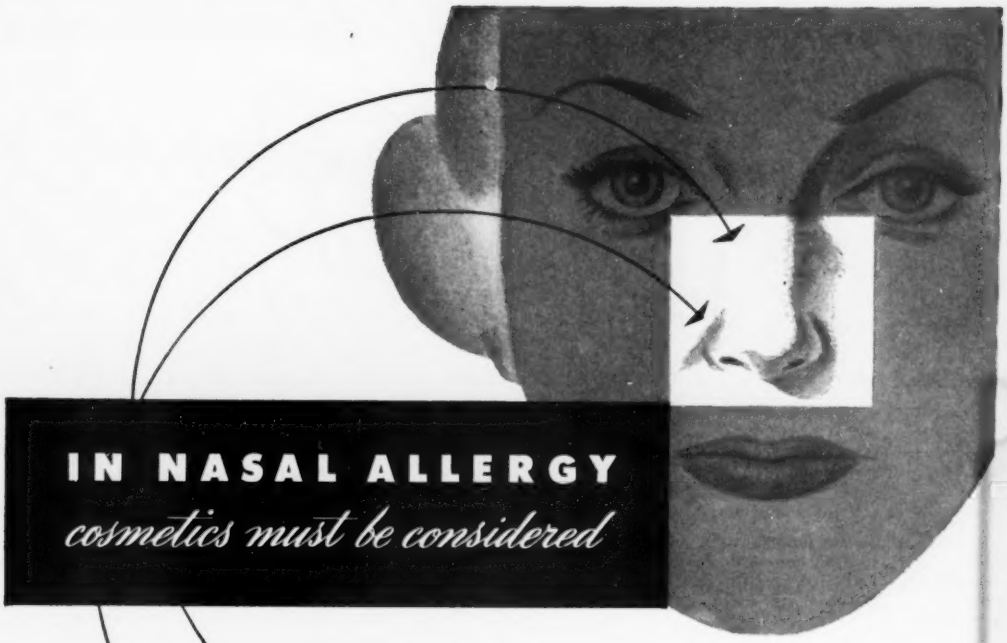

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VOLUME 56 JULY 1952 NUMBER 1

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SUCCESSFUL CLOSURE OF CEREBROSPINAL FLUID RHINORRHEA BY ENDONASAL SURGERY

OSCAR HIRSCH, M.D.
BOSTON

THE DISCHARGE of cerebrospinal fluid through the nose after injury and operation and its spontaneous escape through the nose is known today as a common occurrence. The first description of a cerebrospinal fluid rhinorrhea after fracture of the skull dates back, according to St. Clair Thomson,[1] to Bidloo the Elder, in 1745 (cited by Morgagni). Apparently this had been forgotten, because Blandin [2] in 1840 claimed to be the first to describe such an incident. Spontaneous cerebrospinal rhinorrhea was described by Charles Miller in 1826, according to Thomson and Negus,[3] in a patient with hydrocephalus in whom communications were found at autopsy between the nasal and the cranial cavity.

The observation of an aerocele during lifetime became possible only with the introduction of x-ray techniques. The first diagnosis of this sort was published by Luckett [4] in 1913. There existed only one case, that of H. Chiari [5] in 1884, during the pre-x-ray era, which was diagnosed as aerocele post mortem; this condition had developed during life, and in this case even the communication with the ethmoidal cells was located (Fig. 1).

When the first cases with spontaneous flow of watery fluid from the nose were observed, it was not certain whether this fluid was of cerebrospinal origin or was produced by vasomotor rhinitis. The diagnosis was established by chemical examination. St. Clair Thomson,[1] in his book "The Cerebrospinal Fluid" (1899), described the difference between cerebrospinal rhinorrhea and nasal rhinorrhea.

Cerebrospinal rhinorrhea is of insidious onset, with continuous flow even during sleep. The fluid is clear, like water, and colorless. Its specific gravity is 1.004 to 1.006. Sediment of an alcoholic extract shows needle-like crystals and contains a substance which reduces Fehling's solution but does not ferment with yeast.

Consultant at the Boston City Hospital and Associate in Laryngology at Beth Israel Hospital.

First presented at the Thirty-Fifth Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Victoria, B. C., Canada, May 26, 1951.

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3. Thomson, S., and Negus, V. E.: Diseases of the Nose and Throat, Ed. 5, New York, Appleton-Century-Crofts, Inc., 1947, p. 104.

4. Luckett, W. H.: Air in the Ventricle of the Brain Following Fracture of the Skull, Surg. Gynec. & Obst. **17**:237, 1913.

5. Chiari, H.: Ueber einen Fall von Luftansammlung in den Ventrikeln des menschlichen Gehirns, Heilk. Prag. **5**:383, 1884.

Nasal rhinorrhea (hydrorrhea, vasomotor rhinitis) often sets in with sneezing, with intermissions between attacks and cessation during sleep. The fluid is watery, white, and opalescent, and precipitation with acetic acid indicates the presence of mucin; it contains a substance which reduces Fehling's solution and ferments with yeast.

Thomson mentions also the difference between lymph and cerebrospinal fluid. Lymph coagulates spontaneously, has an opalescent color, and has a much higher

Fig. 1.—Large intravital aerocele discovered post mortem by H. Chiari. Bristles show communications with ethmoidal and nasal cavity through cribriform plate.

specific gravity (1.012 to 1.022); it responds to all tests for sugar and contains albumin.

Identification of cerebrospinal rhinorrhea is readily accomplished by injection of a neutral dye into the subarachnoid space and subsequent recovery of dye in the nasal discharge.

If during a nasal operation a clear watery fluid escapes, there is little doubt that one is dealing with an operative leakage of cerebrospinal fluid. Such mishap may occur when a meningocele is mistaken for a nasal polyp in the cribriform region or when the roof of the nose is fractured during an ethmoid operation. If, during a trans-sphenoidal-pituitary operation, after the incision of the periosteum

of the sella watery fluid escapes, one has to decide immediately whether one is dealing with cerebrospinal fluid or with the contents of a cyst. Both the cerebrospinal fluid and, occasionally, the contents of a cyst may be watery. If aspirated by needle, the fluids differ in color. Cerebrospinal fluid is colorless; the contents of a cyst are yellow, pink, or brown. If cerebrospinal fluid escapes through an operative cut, it may be pink also, because it contains red blood cells. The fluid may stop flowing for some time if the base of the brain has shifted and tamponed the arachnoid-subdural space in the anterior basal cisterns, but the flow of cerebrospinal fluid will continue shortly, possibly when the patient changes his position. The contents of a cyst definitely stop flowing very soon after the cyst has been incised. The cystic fluid contains cholesterol crystals and is therefore glittery and coagulates fast, whereas cerebrospinal fluid is not glittery and does not coagulate. There is no difficulty in recognizing the contents of a cyst as such if they are of an oily consistency. Immediate recognition is of importance because cerebrospinal-fluid leakage has to be dealt with immediately, and the dural opening should not be further enlarged. If a cyst has been opened, the incision has to be enlarged and the edges have to be excised.

NONOPERATIVE TREATMENT OF CEREBROSPINAL-FLUID LEAKAGE

Expectant.—There are a number of observations in the literature that postoperative and spontaneous cerebrospinal rhinorrhea can last for a long period of years or that it can stop spontaneously. Campbell [6] observed a postethmoidectomy rhinorrhea which stopped after two weeks, a not infrequent observation. Thomson and Negus [3] mentioned, in the fifth edition of their textbook, the case of Charles Miller, the first recorded instance of spontaneous cerebrospinal rhinorrhea, starting when the patient was 14½ years of age and continuing until death, at the age of 16½ years. Wurster [7] saw a case of rhinorrhea of six and one-half years' duration, with onset after sneezing, which healed spontaneously and remained closed for a three year period of observation. The Bulsons [8] observed a case of postoperative cerebrospinal rhinorrhea for seven years. One case of spontaneous cerebrospinal rhinorrhea of 10 years' duration was observed by Singleton [9] and another, of 18 years' duration, was observed by Plum.[10] Fribourg-Blanc, Lasalle, and Germain [11] describe a case of post-traumatic cerebrospinal rhinorrhea and pneumatocele of 17 years' duration, with intermittent spontaneous closure for short periods. Calvert [12] states in a discussion of war injuries to the frontal and ethmoidal sinuses that in a number of these cases of cerebrospinal rhinorrhea, in some combined with aerocele, the cerebrospinal rhinorrhea healed spontaneously.

6. Campbell, E. H.: Cerebro-Spinal Rhinorrhea Following Intranasal Surgery, Ann. Otol. Rhin. & Laryng. **37**:865-876, 1928.

7. Wurster, H. C.: Cerebro-Spinal Rhinorrhea: Report of an Unusual Case, J. Indiana M. A. **30**:199-201, 225, 1937.

8. Bulson, A. E., and Bulson, E. L.: Cerebro-Spinal Rhinorrhea Following Intranasal Surgery, J. A. M. A. **93**:1969-1970, 1929.

9. Singleton, A. B.: Leakage of Cerebro-Spinal Fluid Through Cribriform Plate of Ethmoid Bone, Canad. M. A. J. **24**:838-839, 1931.

10. Plum, F. A.: Cerebro-Spinal Rhinorrhea, Arch. Otolaryng. **13**:84-86, 1931.

11. Fribourg-Blanc, Lasalle, and Germain: Deux observations de pneumocèle intracranienne, Rev. neurol. **2**:51-62, 1934.

12. Calvert, C. A.: Discussion on Injuries of the Frontal and Ethmoidal Sinuses, Proc. Roy. Soc. Med. **35**:805-810, 1942.

I operated on a 23-year-old woman on Jan. 13, 1930, for a cystic pituitary adenoma. The patient felt well until May 18, 1948, and obtained a conspicuous improvement in her vision. At that time a gelatinous carcinoma of the nasopharynx was discovered and removed by Dr. Alfred Poelz (Vienna). A leakage of cerebrospinal fluid developed and persisted until May, 1951, when it stopped spontaneously after three years' duration. During this time meningitis developed twice; it was cured promptly by penicillin.

Lumbar Puncture.—In Singleton's [9] case a lumbar puncture was done during an episode of meningitis; after the patient's recovery the nasal dripping stopped. Schroeder [13] mentioned a patient with cerebrospinal rhinorrhea who recovered after treatment with sulfonamides and a lumbar puncture. Frenzel [14] cites a case in which cure was produced by a single lumbar puncture. It is important to reiterate the warning of Grinker and Bucy [15] not to perform lumbar puncture in the presence of cerebrospinal rhinorrhea, as it might cause reflux of infected cerebrospinal fluid.

Cauterization.—B. de Almeida [16] cured a patient with postoperative cerebrospinal rhinorrhea, from whom 650 cc. of cerebrospinal fluid drained daily, by cauterization with 50% chromic acid. The same treatment failed in a second case of spontaneous cerebrospinal rhinorrhea in which the point of escape was cauterized. The patient died of meningitis six months later. In the case of Love and Gay [17] intranasal cauterization applied twice failed to stop the leakage.

X-Ray.—Sgalitzer [18] treated 10 patients with cerebrospinal-fluid leakage after cranial operation. Nine were cured within 2 to 14 days after irradiation, and in one case treatment failed. X-ray treatment was resorted to because of the observation that irradiation of the choroid plexus inhibits the production of cerebrospinal fluid. One patient with cerebrospinal rhinorrhea without demonstrable cause, losing 15 drops a minute, improved after one treatment, and the discharge stopped after the second exposure. The cure lasted two years, until the patient's death due to an intercurrent illness. One patient with cerebrospinal rhinorrhea due to brain tumor was said to have been cured within 14 days by x-ray treatment, but success lasted only three months. A second x-ray treatment failed.

OPERATIVE TREATMENT OF CEREBROSPINAL-FLUID LEAKAGE

Grant,[19] in 1923, was the first to attempt closure of a tear through the dura in a case of traumatic aerocele. The first successful repair of a dural laceration causing

13. Schroeder, M. C.: Meningitis Due to Posttraumatic Cerebro-Spinal Rhinorrhea, Arch. Otolaryng. **40**:206-207, 1944.

14. Frenzel: Discussion, Monatsschr. Ohrenh. **185**:43, 1951.

15. Grinker, R. R., and Bucy, P. C.: Neurology, Springfield, Ill., Charles C Thomas, Publisher, 1949.

16. de Almeida, B.: Zwei Fälle von Kranio-Rhinorrhoe, Monatsschr. Ohrenh. **62**:322-326, 1928.

17. Love, G. J., and Gay, J. R.: Spontaneous Cerebro-Spinal Rhinorrhea: Successful Surgical Treatment, Arch. Otolaryng. **46**:40-44, 1947.

18. Sgalitzer, M.: Erfahrungen mit der Roentgenbehandlung von Liquorfisteln, Wien. med. Wchnschr. **80**:1195-1197, 1930. Sgalitzer also quotes Hover concerning the effectiveness of intravenous injection of hypertonic salt solution.

19. Grant, F. C.: Intracranial Aerocele Following a Fracture of the Skull, Surg. Gynec. & Obst. **36**:251, 1923.

cerebrospinal rhinorrhea was reported by Dandy [20] in 1926. He closed the leakage behind the posterior wall of the frontal sinus by suturing a transplant of fascia lata over the tear. This case is also the first instance of a pneumatocele treated by operative maneuver. Learmouth [21] inserted extradurally over the cribriform plate cotton packs soaked in tincture of iodine and removed them in nine days, with cessation of drainage. Cushing,[22] in 1927, used a transplant of fascia lata as a preventive measure in operations on frontoethmoidal osteomata after two patients had died of meningeal complications. Rand [23] (1930) used suture and muscle. McKinney [24] (1932), Cairns [25] (1937), Gissane and Rank [26] (1940), Eden [27] (1942), and Campbell, Howard, and Weary [28] (1942) also successfully used a piece of fascia for closure of cerebrospinal-fluid leakage. Echols and Holcombe [29] (1941) used pieces of muscle. German [30] (1944) in 5 cases closed fistulae in the cribriform plate by constructing flaps from the dural covering of the crista galli and from the falx. Peet [31] had advocated packing of the wound in traumatic cerebrospinal rhinorrhea, and Gurdjian and Webster [32] have used it, but, according to Dandy, tight closure is preferable. Ecker [33] (1947) used a free periosteal graft over the cribriform plate. Cloward and Cunningham [34] (1947) used absorbable gelatin sponge (gelfoam®). Love and Gay [35] stripped the dura from both sides of the leak in the cribriform plate and sutured the edges, using, in addition, wax and

20. Dandy, W. E.: Pneumocephalus (Intracranial Pneumatocele or Aerocele), Arch. Surg. **12**:949-982, 1926.

21. Learmouth, J. R.: Cerebro-Spinal Rhinorrhea Treated by Operation, Proc. Staff Meet., Mayo Clin. **4**:115, 1929.

22. Cushing, H.: Experiences with Orbito-Ethmoidal Osteomata Having Intracranial Complications, with the Report of Four Cases, Surg. Gynec. & Obst. **44**:721-742, 1927.

23. Rand, C. W.: Traumatic Pneumocephalus: Report of 8 Cases, Arch. Surg. **20**:935-958, 1930.

24. McKinney, R.: Traumatic Pneumocephalus, Ann. Otol. Rhin. & Laryng. **41**:597-600, 1932.

25. Cairns, H.: Injuries of the Frontal and Ethmoidal Sinuses with Special Reference to Cerebro-Spinal Rhinorrhoea and Aeroceles, J. Laryng. & Otol. **52**:589-623, 1937.

26. Gissane, W., and Rank, B. K.: Post-Traumatic Cerebro-Spinal Rhinorrhoea with Case Report, Brit. J. Surg. **27**:717, 1940.

27. Eden, K.: Traumatic Cerebro-Spinal Rhinorrhoea: Repair of a Fistula by a Transfrontal Intradural Operation, Brit. J. Surg. **29**:299-303, 1942.

28. Campbell, E.; Howard, W. P., and Weary, W. B.: Gunshot Wounds of the Brain, Arch. Surg. **44**:789, 1942.

29. Echols, D. H., and Holcombe, R. G.: Traumatic Intra-Cerebral Pneumatocele, South. Surgeon **10**:589-591, 1941.

30. German, W. G.: Cerebro-Spinal Rhinorrhea-Surgical Repair, J. Neurosurg. **1**:60-66, 1944.

31. Peet, M. M.: Symptoms, Diagnosis, and Treatment of Acute Cranial and Intracranial Injuries, New York J. Med. **28**:555-562, 1928.

32. Gurdjian, E. S., and Webster, J. E.: Surgical Management of Compound Depressed Fracture of Frontal Sinus, Cerebro-Spinal Rhinorrhea and Pneumocephalus, Arch. Otolaryng. **39**:287-306, 1944.

33. Ecker, A.: Cerebro-Spinal Rhinorrhea by Way of the Eustachian Tube, J. Neurosurg. **4**:177-180, 1947.

34. Cloward, R. B., and Cunningham, B. E.: The Use of Gelatine Sponge in Prevention and Treatment of Cerebro-Spinal Rhinorrhea, J. Neurosurg. **4**:519-525, 1947.

35. Love, G. J., and Gay, J. R.: Spontaneous Cerebro-Spinal Rhinorrhea: Successful Surgical Treatment, Arch. Otolaryng. **46**:40-44, 1947.

absorbable gelatin sponge. Dandy [36] (1944) published 11 cases of cerebrospinal-fluid leakage, in 2 of which he successfully closed the defect in the bone with wax. This was necessary because after closure of the dura cerebrospinal fluid leaked through the suture line and escaped through the defect in the ethmoidal roof. If the dura is tightly closed and the suture holds, plugging of the bone is usually not necessary. Adson [37] recommended bilateral exploratory operation, if necessary, to determine the size of the opening. Graham [37a] reports a case of a spontaneous cerebrospinal rhinorrhea in which the openings of the cribriform plate were successfully filled with wax. Coleman [37b] operated on patients with traumatic cerebrospinal rhinorrhea and pneumatocele by releasing the air and suturing the dura over the cribriform plate.

The interest of rhinologists in cerebrospinal-fluid leakage was aroused by the numerous head injuries during the World Wars, especially during World War II. There was a discussion on this subject in the combined session of the rhinologists and neurosurgeons in the laryngological section of the Royal Society of Medicine (July 17, 1942). C. A. Calvert [12] reported for the rhinologists and Hugh Cairns for the neurosurgeons. In a series of 1,731 cases of head injury, there were 128 fractures of the frontal and ethmoidal sinuses. Among these were (1) 21 cases with cerebrospinal rhinorrhea, (2) 9 cases of intracranial aerocele, and (3) 13 cases of no cerebrospinal rhinorrhea and no aerocele. The presence of dural laceration was either demonstrated at operation or proved to have been present in patients who succumbed to meningitis. Operations were performed because of fractures visible on x-rays.

The three groups provide a total of 43 cases with positive evidence of dural tear complicating frontal and ethmoidal sinus fractures. In 20 cases the patient was treated conservatively; 9 patients recovered without incident, and either meningitis or brain abscess developed in 11, of whom 6 died. In 23 cases the patient underwent a dural repair operation; 20 patients recovered without incident, and 3 died.

The joint conclusion was that there is some risk in withholding dural repair in cases of cerebrospinal-fluid leakage, even if the leakage has been slight or has ceased within a few days. Meningitis or brain abscess has developed in patients several months later.

A similar conclusion was reached at the joint session of the Austrian Otolaryngologists and Neurosurgeons at the Sixth Otolaryngological Meeting, in 1950.[38] The neurosurgeons advocated closure of the leakage by fascial graft and obliteration of the frontal sinus in cases due to injury of the posterior (cerebral) and orbital walls of the frontal sinus and adjacent dura. The rhinologists pleaded for an operation as radical as necessary but as conservative as possible.[39]

Dohlman described an instance of repair of a spontaneous cerebrospinal rhinorrhea from the cribriform region by means of a flap taken from the middle turbinate.

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36. Dandy, W. E.: Treatment of Rhinorrhea and Otorrhea, Arch. Surg. **49**:75-85, 1944.

37. Adson, A. W.: Cerebro-Spinal Rhinorrhea, Ann. Surg. **114**:697, 1941.

37a. Graham, T. O.: Cerebro-Spinal Rhinorrhea, J. Laryng. & Otol. **52**:344, 1937.

37b. Coleman, C. C.: Fracture of the Skull Involving Paranasal Sinuses and Mastoids, J. A. M. A. **109**:1613-1616, 1937.

38. VI. Oesterreichischer Oto-Laryngologen-Tag, Monatsschr. Ohrenh. **185**:37-45, 1951.

39. Dohlman, G.: Spontaneous Cerebro-Spinal Rhinorrhea: Case Operated by Rhinologic Methods, Acta Oto-laryng. Supp. 67, pp. 20-23, 1948.

Cerebrospinal rhinorrhea is not uncommon. The incidence of spontaneous air in the cranial cavity is less frequent. Horrax [40] (1921) found seven cases in the literature. Dandy [20] (1926) was able to gather 25 cases, but Plunkett and Lendrum [41] (1935) collected 101 cases of pneumatocele. Dandy found the mortality in this situation, if untreated, to be 40% ; Plunkett and Lendrum, in cases with and without treatment found it to be 30%.

My interest in the urgent necessity for closure of a cerebrospinal fistula originated when I happened to produce one in the course of an operation. The operation was a sella decompression, a procedure originally suggested by A. Schueller,[42] who noticed that the intracranial pressure, trepanation, often leads to widening and thinning of the floor of the sella.

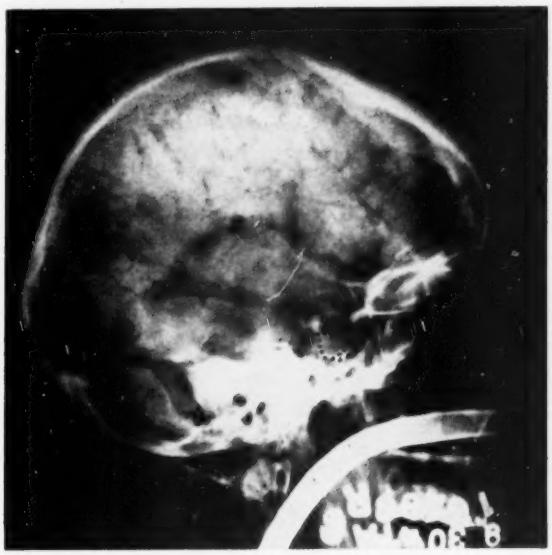

Fig. 2.—Pneumoencephalogram. Dotted convex lines correspond to double-contoured deep sella; horizontal slightly concave line indicates diaphragm which by dilated third ventricle is pushed under original level, i. e., horizontal—slightly concave—between anterior and posterior clinoid processes.

B. B., a 39-year-old woman, suffered from terrific headache since 1945, allegedly after a fall on the back. She was married at the age of 16 and had one child. She had had a hysterectomy at age 25. She was hospitalized eight times because of terrific headache, treated by x-ray and drugs, and became addicted to morphine. The x-ray showed an abnormally deep sella. No symptoms or signs of a pituitary tumor were elicited by observation and examination, including an examination of vision. The pneumoencephalogram revealed that the third ventricle reached under the level of the diaphragm (Fig. 2). March 28, 1947, a sellar decompression was agreed upon in an effort to relieve intracranial pressure. I opened the bony floor of

40. Horrax, G.: Intracranial Aerocele Following Fractured Skull, Ann. Surg. **73**:18-22, 1921.

41. Plunkett, J. E., and Lendrum, F. C.: A Case of Spontaneous Pneumocephalus and Cerebro-Spinal Rhinorrhea, Canad. M. A. J. **33**:512, 1935.

42. Schueller, A.: Sellare Paliativtrepanations, Wien. med. Wchnschr. **61**:182-190, 1911.

the widened sella by chisel and removed it by punches to a diameter of 1.75 cm. longitudinally and 1.5 cm. laterally. Needle puncture through the periosteum of the sella did not reveal fluid. The incision was made in the posterior part. Fluid poured from the anteriormost part of this incision, but after a minute the flow stopped; hence I had the impression I had opened a cyst. I finished the operation by putting strips of petrolatum gauze on both sides of the septum. The first day the patient had no headache. She felt well until March 31. Leakage of cerebrospinal fluid was proved. Therefore a piece of fascia was placed endonasally over the leakage within the sphenoidal cavity, but without success. On April 2, Dr. H. Hamlin performed a craniotomy, located the leakage in the anterior part of the sella, and applied a piece of fascia covered by absorbable gelatin sponge. The leakage stopped. The patient was in good condition for one week. On April 11 her temperature rose to 103 F. A lumbar puncture showed opaque fluid. Aureomycin and penicillin were given parenterally. On April 15 the patient was in good condition with a normal temperature and no cerebrospinal-fluid leakage. Lumbar puncture yielded normal fluid. There was no recurrence up to December, 1951.

This procedure is similar technically and in its success to the method used by Cushing in closing cribriform leakage. The opening which had to be closed in this case was situated in the anterior part of the sella. Owing to the postfixed chiasm it was, fortunately, possible to gain access to the floor of the sella. This appears to be the first operative closure of a leakage within the floor of the sella turcica by an intracranial approach.

ENDONASAL CLOSURE OF CEREBROSPINAL LEAKAGE

The fistulae in the following two cases presented a different problem because the leakage was situated in a spot which could not be reached by intracranial approach without endangering the patient's life. This spot was in the posterior surface of the dorsum sellae at its junction with the clivus, located dorsal to the infundibulum. Transfrontal intervention was inapplicable because the site of the leakage could be reached only by damaging the infundibulum, which would produce diabetes insipidus; possible manipulation in the neighborhood of the hypothalamus would invite greater disaster. Therefore it was necessary to devise a less risky method.

Case 1.—P. S., a 32-year-old female patient of Dr. J. C. Fox (Hartford, Conn.) and Dr. H. Hamlin (Providence, R. I.), was admitted to the Rhode Island Hospital Aug. 19, 1949, because of progressive acromegaly, headache, and visual disturbance. She began to have headaches approximately 10 years before admission, which became more troublesome and worse following the birth of her only child six years previously. She was treated at various clinics for migraine and suspected allergic conditions following her pregnancy. There was considerable weight gain, overgrowth of hands, broadening and coarsening of facial features, and also increased appetite. The patient stated that she often had to eat six times a day, even during the night. Her voice became deep and her skin thick. Menstruation reappeared six months after the birth of her child but at irregular intervals. Her last period had been in July, 1949. The patient stated that she was able to see well but that her field of vision had been encroached upon from the upper and outer sides as if she had blinders on. A diagnosis of acromegaly and suspected acidophile pituitary tumor was made by Dr. J. C. Fox, and she was given x-ray therapy, without improvement.

Examination.—The patient was a well-developed, obese woman in no distress, and was alert and cooperative. She had a low-pitched voice and obvious acromegalic features. Her skin was coarse, her thorax was broad and barrel-shaped, and her breasts were large and heavy. Hair distribution was normal. Water displacement of the right hand to the tips of the radius and ulna was 425 cc., and of the left hand 420 cc. She wore size 7 gloves 10 years ago and now wears size 8. The size of her shoes changed from 8½-A to 9½-C. Exami-

nation of her visual fields with a 1/1,000 test object showed a temporal and upper field defect on the right side and on the left side a defect of both upper quadrants to the horizontal which reached 5 degrees, simulating altitudinal hemianopsia. An x-ray of the sella showed moderate but definite enlargement. The anterior clinoid processes were of normal shape; the dorsum of the sella reclined to the vertical position and there was calcification in the region of the sella. Operation was advised because of the visual field restriction.

A trans-sphenoidal-septal operation was done Aug. 20, 1949, with the patient under endotracheal ether anesthesia. To prevent bleeding, the septum was infiltrated bilaterally with 1% procaine hydrochloride plus epinephrine. Submucous resection was carried out as far as the anterior wall of the sphenoidal sinuses, which were easily exposed, infracted, and removed. The intersphenoidal septum was about 1 cm. long and was removed also. The sinuses were asymmetric, the right being narrower than the left. The sella was opened and enlarged by special burrs in all directions. After incision of the exposed periosteum of the sella, yellowish serous fluid appeared. The first impression was that the fluid was cerebrospinal fluid and then the flow stopped. Because of the calcification I thought it might be a cyst, and I enlarged the incision from 2 to 3 mm. More fluid, of the same type, drained. Therefore, I laid absorbable gelatin sponge over the incision, put the flaps of the septum together, and finished the operation by applying petrolatum tampons to both sides of the septum. The patient was maintained on penicillin and dihydrostreptomycin.

On Aug. 21 the patient had a headache during the night but was better the next day; there had been no vomiting since the operation, although she had taken fluid and food. On Aug. 22 the petrolatum tampons were removed; the patient's temperature was 99.4 to 99.6 F., and her pulse rate was 78. On Aug. 23 dripping of cerebrospinal fluid was noted. Cotton plugs had to be removed three times during the afternoon. The patient's temperature was 99.8 F., and she had vomited.

The flaps of the septum were separated and the sphenoidal cavities exposed. To the right of the midline a pulsating point was visualized; absorbable gelatin sponge soaked with thrombin was applied, the whole cavity was filled with pieces of absorbable gelatin sponge, and the septal flaps were put together. On Sept. 15 leakage was noted when the patient turned on her side. Her temperature was normal; her pulse rate was 94.

On Sept. 28 the septal flaps were separated and the sphenoidal sinuses exposed. A skin graft covered with absorbable gelatin sponge was applied to the pulsating point within the sphenoidal sinus. Leakage ceased for several days.

On Oct. 29 the cerebrospinal fluid drip again became continuous, especially on the patient's changing position. It was decided to reopen and review the field of operation. The flaps of the septum were separated and the sphenoidal sinuses reached. The patient vomited several times and so the operation had to be abandoned, but oxidized cellulose (oxycel®) was packed against the floor of the sella.

An x-ray showed an aerocele above the dorsum of the sella surrounding the pituitary stalk (Fig. 3).

On Nov. 12 the leaking was still recurrent. Aerobacter aerogenes was cultured from the cerebrospinal fluid. On Nov. 18 there were no bacteria in the cerebrospinal fluid.

On November 26 the flaps of the septum were separated again. The sella was opened in its anterior part, and its bone was removed. The exposed periosteum was cut into a U-shape and turned back over the fistula. The pituitary tumor presented itself as a brownish-red body and was removed. On microscopical examination it proved to be an eosinophile tumor. The flap formed from the dura contracted immediately and proved to be of inadequate size. Therefore another flap of mucoperichondrium was dissected from the posterior part of the septum and was turned into the sphenoidal cavity toward the floor of the sella and pressed against the fistula with oxidized cellulose gauze and rubber tampon. The leakage diminished for a short time but recurred as usual.

On Dec. 4, with the patient under local anesthesia, the field of operation was checked: The flap had shrunk away from the sella. A new flap, comprising three-fourths of the length of the septum, was raised from the opposite side and turned with its raw surface into the sphenoidal cavity along the floor of the sella and the posterior walls of the sinus (Figs. 4 and 5). It was long enough to cover the whole floor with allowance for shrinkage. It was held in this position by a rubber-covered gauze tampon.

On Dec. 10 slight leakage was still present; therefore another revision of the operative field was done with the area under local anesthesia. The flap was intact but had curled at its end. The flap was straightened. The floor of the sella and the flap itself were painted with 50% silver nitrate solution and held in the straightened position by a small piece of petrolatum gauze. After each operative procedure the patient was given antibiotics: penicillin, aureomycin, and dihydrostreptomycin.

During the week of Dec. 10 to 17 nasal dripping was definitely less. Inspection of the field on Dec. 17 revealed the flap to be in correct position. Leakage stopped definitely on Dec. 23 and has not recurred since (two years after final operation). There was some improvement of headache and bulimia; hand measurements were reduced.

Summary.—A 32-year-old acromegalic woman was operated on transsphenoidally because of visual disturbance and headache due to a pituitary tumor. A cerebrospinal-fluid leakage was produced at the end of the operation in the region

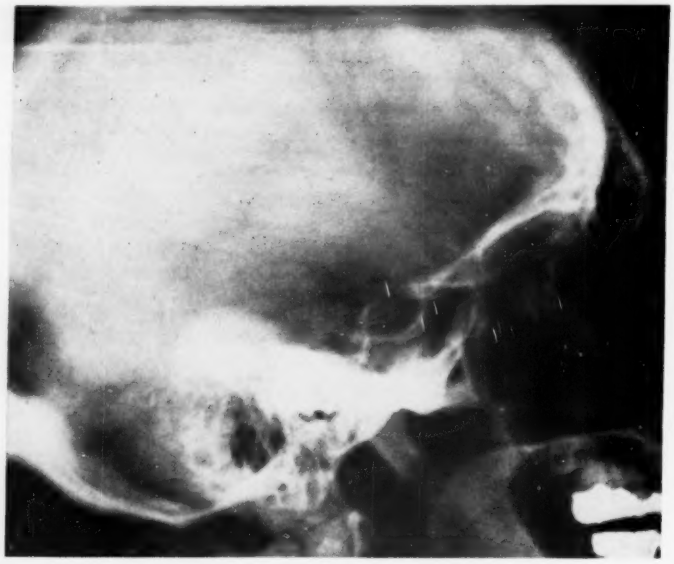

Fig. 3.—Postoperative aerocele surrounding pituitary stalk.

of the junction of the dorsum sellae with the sphenoidal cavity. This location beyond the infundibulum was not accessible by intracranial approach. Repeated treatment by application of absorbable gelatin sponge, skin graft, and periosteum of the sella failed to close the fistula. Finally a long flap was cut out of the septum and turned with its raw surface into the sphenoidal cavity and maintained in this position by a petrolatum tampon. It was readjusted when its end had curled. This procedure was followed by closure of the fistula. A defect in the nasal septum resulted which produced no discomfort. Since this procedure was carried out in the fourth month after the initial operative complication, when the area around the leakage had healed, effort was made to stimulate these tissues and the donor surface of the flap by application of 50% silver nitrate solution.

Case 2.—A. Z., a 32-year-old white woman, a patient of Dr. F. Bartter (Massachusetts General Hospital), suffered from acromegaly with headache and papilledema. Acuity of

vision and field of vision were normal. Menstruation was irregular. The sella was definitely but moderately enlarged. On June 6, 1950, a trans-sphenoidal-septal operation was performed with the patient under general anesthesia. The anterior walls of the sphenoidal cavities were thick and were opened by chisel. The right sphenoidal sinus was much narrower than the

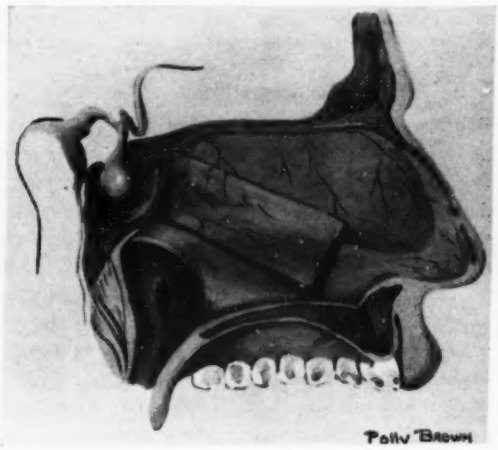

Fig. 4.—Contours of flap raised from septum, long enough to reach leakage and allow shrinkage.

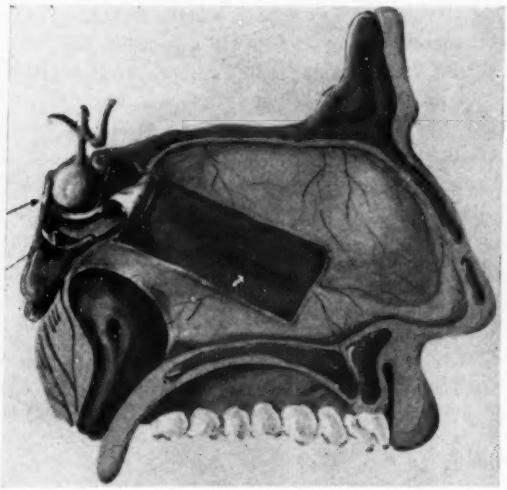

Fig. 5.—Flap bent into sphenoidal cavity, covering by its raw surface site of leakage. Upper arrow points to site of leakage; lower arrow points to end of flap.

left. The enlarged sella became visible and also had to be opened by chisel. The dura was incised. No solid resistance was encountered, and while the curette was being used cerebrospinal-fluid leakage occurred. I finished the operation by covering the fistula with a flap from the septum and a petrolatum tampon to hold the flap in position. The leakage of cerebro-

spinal fluid stopped. The patient felt well; the rectal temperature was 100.4 F. After removal of the tampon on June 12, leakage was again apparent. On June 15 the patient's temperature was 100.8 F. and lumbar puncture revealed cloudy fluid. She was treated by penicillin and streptomycin intrathecally. On June 17 her temperature was normal and lumbar puncture yielded clear fluid. On June 18 bleeding from the nose was controlled by oxidized cellulose. On June 21 cerebrospinal fluid leakage ceased. Streptomycin intrathecally was continued because Pseudomonas aeruginosa (Bacillus pyocyaneus) was cultured from the first lumbar puncture. She was kept in the hospital under treatment for six weeks.

COMMENT

Two patients with acromegaly, similar in age and symptoms and in the configuration of the moderately widened sella, were operated on by the trans-sphenoidal-septal method. The indication in the first case was constricted visual fields and in the second case papilledema. Both patients suffered from intractable headache. In both cases the sphenoidal sinuses were asymmetric and leakage of cerebrospinal fluid was produced at points which could not be reached by transfrontal approach, demanding an attempt to close the fistula endonasally. The site of the leakage was exposed and successfully closed by a hitherto undescribed technique.

The procedure used in both cases is shown in Figures 4 and 5. A submucous septal resection had been done; therefore the septum consisted of two mucoperichondrial flaps. These were separated in the first case after four months of healing. In this case two attempts were made to close the leakage. On the first attempt the flap used for the closure of the leakage was too short and the result unsatisfactory. On the second effort the flap was three-fourths of the length of the septum and was turned with its raw surface into the sphenoid sinus and against the fistula. This raw surface of the second flap was in a state of healing because it had been dissected from the corresponding and previously used flap. Therefore its raw surface was stimulated by application of 50% silver nitrate solution, which was also applied in the region of the fistula. Since mucosal flaps from both sides of the septum had to be used, a defect in the septum was produced, which, fortunately, is of no discomfort to the patient.

In the second case the fistula was repaired immediately. The flap, as well as the region of the leakage, was fresh and consequently healing union resulted and the leakage stopped in due course. In both cases bacterial meningitis developed which was cured by antibiotic therapy. In both cases the fistulae were within the sphenoidal cavity, but one can suggest that the procedure described may prove to be a basis for closure of cerebrospinal-fluid leakage in any part of the nose.

SUMMARY

The literature on cerebrospinal rhinorrhea and aerocele of traumatic and spontaneous origin is reviewed.

Differences among cerebrospinal fluid, vasomotor rhinorrhea, nasal lymphorrhea, and contents of cysts in the sellar region are recalled.

Various kinds of treatments of cerebrospinal-fluid leakage are cited, including an instance of operative intrasellar fistula repaired by transfrontal approach.

A new procedure, by which operative cerebrospinal-fluid leakage within the sphenoidal sinus was successfully closed endonasally in two patients, is described and illustrated.

186 Commonwealth Ave. (16).

TOPICAL ANESTHESIA IN BRONCHOSCOPY

HENRY J. RUBIN, M.D.
AND
BARNEY M. KULLY, M.D.
LOS ANGELES

THERE is little in the matter of topical anesthesia about which endoscopists are in unanimous accord. This is emphasized by Jackson and McReynolds [1] and Himalstein,[2] who sent questionnaires to members of the American Broncho-Esophagological Association. Replies revealed wide differences of opinion about such basic considerations as choice of drug, concentrations, and amounts; preliminary testing for sensitivity, and the use of sprays, pyriform sinus applicators, and epinephrine. There is agreement only on the importance of preliminary sedation and slow administration. That so many persons of experience employ with continued success such varying methods suggests that there are a number of techniques of equal worth and that it is presumptuous for one endoscopist to regard his as superior to others which have proved practical. Taking cognizance of this, we present our procedure for anesthesia, pointing out only that an effort has been made to base it upon certain experimental and clinical evidence.

TOXICITY

Of the many topical anesthetics which have been popular at one time or another, only two, cocaine and tetracaine (pontocaine®), remain in general use. Both are the subject of occasional reviews based on case reports describing toxic reactions.

Interest in the toxicity of cocaine was keener 25 years ago than now, and numerous investigations culminated in the report of the Mayer Committee [3] of the American Medical Association in 1924. This study listed and evaluated all reported accidents and made specific recommendations as to dosage and concentrations. It is questionable whether this had any appreciable effect on the techniques in general use, because the dose recommended as being safe was so small (a total of not more than 1½ grains [90 mg.] as to be of little practical worth. Consideration was not given to the avoidance of reactions due to hypersensitivity.

From the Department of Tuberculosis, Los Angeles County General Hospital, and the Department of Otolaryngology, Cedars of Lebanon Hospital.

1. Jackson, C. L., and McReynolds, G. S.: Anesthesia for Peroral Endoscopy, Ann. Otol., Rhin. & Laryng. **49**:1048, 1940.

2. Himalstein, M. R.: Topical Anesthesia in Endoscopy: Critical Evaluation with Case Reports and Survey of Current Opinion, Laryngoscope **59**:1102, 1949.

3. Mayer, E.: The Toxic Effects Following the Use of Local Anesthetics: An Analysis of the Reports of 43 Deaths Submitted to the Committee for the Study of Toxic Effects of Local Anesthetics of the American Medical Association, and the Recommendations of the Committee, J. A. M. A. **82**:876 (March 15) 1924.

Tetracaine was synthesized in 1929, and reports have appeared from time to time on its toxicity. There have been no official recommendations relating to its use other than those of the manufacturer.

Toxic reactions to these drugs are due to overdosage, hypersensitivity, or too rapid administration. Despite these limited causes for reaction it is frequently difficult to ascertain from reports of accidents which one of these factors is responsible in a given case. Commonly read are remarks such as, "In retrospect, more than 4 cc. may have been given," "The exact quantity is unknown but is believed to be . . .," "The amount was roughly gauged to be . . .," etc. In these instances the most important single thing to be known, namely total dosage in grains or milligrams, can only be conjectured. If the total dose happens to have been recorded, another important factor may have been omitted, such as the length of time during which the anesthetic was injected into the tracheobronchial tree. Accidents strike with such tragic swiftness that certain of these salient details are often obscured in the excitement. There is no doubt that reactions and deaths due to bona fide sensitivity also occur, but one may reasonably conclude from the published reports that many accidents presumably due to hypersensitivity are in fact the result of inadvertent overdosage. This has been stressed by numerous authors.[4]

That overdosage is the chief cause of reactions, particularly in the case of tetracaine, is suggested by the infrequency with which they occur when this drug is injected as a regional (other than spinal) anesthetic. Bonica[5] recently reported 3,000 cases without fatality, and he lists an extensive bibliography. He remarks, "Reports have appeared in the literature regarding severe toxic reactions and fatalities which have occurred during topical application of pontocaine, but none may be found during infiltration and block anesthesia." It may be countered that absorption is more rapid from mucous membranes, and this is true. It was demonstrated experimentally by one of us (H. J. R.[6]) that the minimum lethal dose of tetracaine is four times as great by subcutaneous injection as by instillation into the tracheobronchial tree. However, anaphylactic shock is not necessarily related to dosage, and if true hypersensitivity is a significant factor in severe reactions, it should occur to at least some degree in anesthesia by injection. Such reactions, however, are conspicuously rare. It would appear that overdosage is the chief cause of toxic reactions and that close attention to the total dose as well as to speed of administration and preliminary testing for sensitivity would greatly reduce severe reactions and deaths.

4. (*a*) Cullen, S. C.: Anesthesia in General Practice, Chicago, The Year Book Publishers, Inc., 1946, p. 157. (*b*) Titche, L. L.: Anesthesia in Tracheo-Bronchial Procedures, Dis. Chest **13**:452, 1947. (*c*) Field, C. E.: Amethocaine Hydrochloride, Correspondence, Brit. M. J. **1**:236, 1949. (*d*) Seevers, M. H.: Symposium: Anesthesia in Otolaryngologic Surgery; The Preparation of the Patient, Tr. Am. Acad. Ophth. **53**:281 (March-April) 1949. (*e*) Carabelli, A.: Laryngo-Tracheo-Bronchial Anesthesia for Bronchoscopy and Bronchography with Pontocaine, Dis. Chest **15**:532, 1949.

5. Bonica, J. J.: Use of Pontocaine for Regional Anesthesia: Conclusion, Current Res. in Anesth. & Analg. **30**:76, 1951.

6. Rubin, H. J.: Toxicity of Certain Topical Anesthetics Used in Otolaryngology, A. M. A. Arch. Otolaryng. **53**:411 (April) 1951.

TOTAL DOSAGE

Specific doses not to be exceeded in topical application have been suggested by the Mayer Committee for cocaine and by the manufacturer for tetracaine. Evidence upon which these recommendations rest turns out upon close scrutiny to be open to question. In the case of tetracaine, the manufacturer urges that 20 mg., which is the equivalent of 1 cc. of a 2% solution, not be exceeded. One would presume that such a positive recommendation was based on reasonably strong evidence, but this is not the case. Bieter [7] noted in 1936, while studying the literature on lethal doses of the local anesthetics in animals, that total safe doses of cocaine and butacaine sulfate (butyn®), as recommended by the Local Anesthetic Committee of the American Medical Association [3] were similar to minimum lethal doses per kilogram of guinea pig administered by subcutaneous injection. It was his opinion that in the complete absence of definite criteria for determining safe human doses the minimum lethal doses per kilogram of guinea pig should be considered as total safe human doses. The figure for tetracaine was 20 mg., and that is the amount which has ever since been held to be the safe maximum. Since 1936, tetracaine has come into its own as both a regional and a topical anesthetic, and there is much evidence [8] that the recommended dose is considered to be too low, that is, too far below what the human organism will safely tolerate, to be practical. That most endoscopists find the recommended maximum dose too little for satisfactory anesthesia is reflected particularly in replies to the recent questionnaire [2] sent to members of the Broncho-Esophagological Association. We reluctantly confess to being unable to obtain satisfactory anesthesia for bronchoscopy in the majority of cases with a total maximum dose of 20 mg., despite reportedly successful techniques,[9] and have for several years been using 50 mg. on all adults without incident. Failure of the manufacturer to recognize this general sentiment regarding dosage is a disservice in that it encourages clinicians to disregard the recommendations entirely in an effort to achieve satisfactory anesthesia. The results are reflected in the ever-increasing literature on the toxicity of tetracaine topically applied.

The situation with cocaine is much the same. In 1924, the Mayer Committee concluded that 1 to 1½ grains (60 to 90 mg.) could be considered a safe amount for topical anesthesia. This is the equivalent of 1 to 2 cc. of a 5% solution, which is very little with which to work. No distinction was drawn between the dose which was presumably completely safe and one which was practical. It might be illustrated analogously that 1 grain of acetylsalicylic acid is a safe dose, one from which severe reactions are not likely to occur. However, the clinician is greatly aided by being officially advised that the practical therapeutic dose of acetylsalicylic acid is 5 grains (300 mg.), and he prescribes accordingly. No such practical recommendation has been forthcoming in the case of cocaine, with the result that,

7. Bieter, R. N.: Applied Pharmacology of Local Anesthetics, Am. J. Surg. **34**:500, 1936.

8. Himalstein.[2] Titche.[4b] Carabelli.[4e] Jackson, C. A.: Amethocaine Hydrochloride, Brit. M. J. **1**:99, 1949. Wilton, T. N. P.: Amethocaine Hydrochloride, Correspondence, Brit. M. J. **1**:235, 1949. Bourne, W. A.: Amethocaine Hydrochloride, Correspondence, Brit. M. J. **1**:367, 1949.

9. Carabelli.[4e] Miller, J.; Mann, F., and Abramson, H.: A Method for Topical Anesthesia by Nebulization of Local Anesthetics, Dis. Chest **16**:408, 1949.

since the officially sanctioned safe dose is too low for complete anesthesia, each endoscopist must determine for himself what constitutes a practical as well as a safe amount.

In an effort to find some reasonable basis for such a dose, one of us (H. J. R.[8]) observed that the toxicity ratio of tetracaine to cocaine, when applied experimentally to the tracheobronchial tree of rabbits, was 6:1. It will be noted that this ratio is more unfavorable to tetracaine than the generally accepted toxicity ratio of 2.5-3:1, which was established by subcutaneous injection.[10] In other words, six times as much cocaine as tetracaine was tolerated intratracheally. Having found 50 mg. of tetracaine (equivalent to 10 cc. of 0.5% solution) adequate clinically, we adopted six times this, or 300 mg. (5 grains), as the adult dose for cocaine and have found it to be completely satisfactory. This is equivalent to 6 cc. of a 5% solution. These amounts are never exceeded in any patient.

CONCENTRATION OF SOLUTION

The concentration of the anesthetic solution has been a controversial subject for years. Experimental evidence has heretofore been limited to conclusions drawn from parenteral administration. Braun[11] demonstrated, as long ago as 1903, that dilution of injected anesthetics decreased their toxicity. The reason for this is clear. When an anesthetic is injected subcutaneously or intramuscularly, it pools at that point, and absorption is limited to its periphery. It follows that the greater the dilution, that is, the larger the fluid mass, the slower must be the absorption. No similar experiments were conducted topically until recently when one of us (H. J. R.[8]) applied cocaine and tetracaine in various concentrations to the mucous membranes of rabbits and guinea pigs. It was found, in contrast to the results by injection, that with speed of administration constant, there was not the least difference in toxicity of various concentrations, the minimal lethal doses being the same, regardless of concentration. The only important factor was total dose of drug. The explanation is apparent. When anesthetic solutions are applied topically to the tracheobronchial mucous membranes, there is no opportunity for pooling as in the case of parenteral injection. Whatever is instilled diffuses immediately because of gravitational pull, and all of the solution contacts absorbing surfaces almost simultaneously.

The practical point to be gathered from this is that the significance of dilute versus concentrated solutions has been exaggerated. The most important single factor in topical anesthesia is not concentration or dilution but rather the total dose.

Concentrated solutions are inferior to dilute solutions, however, for other reasons. First, greater volume of dilute solutions allows contact with a larger area of the tracheobronchial tree, thus affording more complete anesthesia. Second, in case of evaporation by occasional failure to cap stock bottles, hazard to the patient is directly proportional to the strength of the original solution. Third, and

10. Fussgänger, R., and Schaumann, O.: Über ein neues Lokalanästhetikum der Novokainreihe (Pantokain), Arch. exper. Path. u. Pharmakol. **160**:53, 1931.

11. Braun, H.: Über den Einfluss der Vitalität der Gewebe auf die örtlichen und allgemeinen Giftwirkungen localanästhesirender Mittel und über die Bedeutung des Adrenalins für die Localanästhesis, Arch. klin. Chir. **69**:541, 1903.

most important of all, endoscopists who do not specifically measure the amount of anesthetic used for their patients are more likely to exceed safe dosage when using concentrates, because of the very smallness of the amount. The greatest danger lies in the tendency of the physician to forget that the solution he is using so freely is in fact concentrated.

We have found that tetracaine 0.5% and its anesthetic equivalent, cocaine 5%, combine satisfactory anesthesia with ample volume.

SPEED OF ADMINISTRATION

Moore, in discussing a paper by Thomas and Fenton,[12] made the following cogent observation.

> Although a sensitized individual will react to small quantities of the specific allergen, I cannot help but feel that the quantity and method of injecting intratracheally may be very important. If a fair amount is injected in one quick shot, it may all go down one bronchus and into an alveolus. If this occurs, it is absorbed from the alveolus very quickly, and the effect on the patient is the same as though a like quantity had been given intravenously.

We [13] have recently demonstrated experimentally the truth of this remark. Cocaine and tetracaine were instilled into the tracheobronchial tree of rabbits over different periods of time, and it was found that minimum lethal doses were decreased with rapid administration, such as within 15 seconds. Very rapid administration of a quantity sufficient to drop through the bronchi into the alveoli permits more rapid absorption, with consequent unduly elevated blood levels. Shumacker [14] showed that death from excessive local anesthesia occurs when a certain concentration of drug is reached in the blood stream and that the level is the same, regardless of the mode of administration. A rapid intravenous injection results in attaining the critical level quickly, and hence less drug is necessary to cause death than when a slower means of administration is used. Many times the rapid minimum lethal dose may be given, for example, subcutaneously, because slow absorption delays reaching of lethal concentrations in the blood stream. Hence emptying of more than fractional amounts of anesthetic solution at a time into the tracheobronchial tree is to be avoided. What percentage of accidents is due to transgression of this basic principle cannot even be conjectured.

When, however, intratracheal instillation is fractionated over a somewhat longer period, such as 2 minutes, minimum lethal doses are the same as when it is fractionated over 10 minutes. The reason is that both these time periods are sufficiently longer than circulation time to preclude acute rises in blood levels. As emphasized by us [13] previously, this is not to be construed as suggesting that it is safe to complete anesthesia in a short period, nor does it mean that more solution can be used if a longer time is taken. Detoxication of these drugs is so slow [15] that essentially the same amount of unmetabolized anesthetic is present in the body

12. Thomas, J., and Fenton, M.: Fatalities and Constitutional Reactions Following the Use of Pontocaine, J. Allergy **14**:145, 1943.

13. Rubin, H. J., and Kully, B. M.: Speed of Administration as Related to the Toxicity of Certain Topical Anesthetics, Ann. Otol. Rhin. & Laryng., to be published.

14. Shumacker, H. B., Jr.: Reactions to Local Anesthetic Agents: Experimental Studies with Procaine, Surgery **10**:119, 1941.

15. Eggleston, C., and Hatcher, R.: A Further Contribution to the Pharmacology of the Local Anesthetics, J. Pharmacol. & Exper. Therap. **13**:433, 1919.

at the end of 10 minutes as in 2, and additional amounts cannot be safely given. Furthermore, fractionation over 10 minutes, as opposed to 2 or 3 minutes, affords additional protection to hypersensitive individuals. It allows time for a possible reaction to occur with infinitely greater assurance that recovery will ensue than if the entire amount had been given more quickly and irretrievably.

HYPERSENSITIVITY

Untoward reactions to topical anesthetics, not related to overdosage or rapid administration, are admittedly manifestations of undue sensitivity. Which term or terms—idiosyncrasy, hyperergy, allergy, or hypersensitivity—best describe these states is unsettled. From a practical standpoint, the important fact is that persons with such conditions experience reactions of varying severity to quantities of anesthetic which are known to be well tolerated by the average patient.

Various methods of detecting these potential anesthetic casualties have been suggested. Thomas and Fenton [12] recommended patch tests in persons with allergic histories. A Veterans Administration letter [16] advised that small amounts of tetracaine be applied to restricted areas of mucous membrane prior to its use for topical anesthesia. A Veterans Administration Bulletin [17] recommended intradermal testing for sensitivity when indicated. Preliminary sprays, followed by a waiting period before anesthesia is continued, are in general use.

Although the markedly allergic individual would probably do well to have a preliminary skin test of some type, this method of detecting sensitivity is not routinely applicable to a busy endoscopic clinic. Furthermore, as indicated by Thomas and Fenton,[12] the reliability of skin tests is questionable. Derbes and Engelhardt [18] stated:

> It is unfortunate that skin tests with drugs, which would be of great aid in helping avoid these reactions, are of questionable value. While patch tests to drugs causing skin dermatitis are highly specific and reliable, drugs which cause trouble following parenteral administration often cannot be satisfactorily tested either by patch tests or scratch tests or intracutaneous tests.

We feel that direct application to the oral or pharyngeal mucous membranes is preferable. It has been demonstrated experimentally [6] that absorption of cocaine and tetracaine is slower when administered orally than when instilled into the nose or tracheobronchial tree. This is a factor making preliminary administration to the mouth or throat more suitable than to the nasal or tracheobronchial membranes.

A preliminary spray has the great disadvantage of delivering an unknown quantity of solution. Not only does one atomizer differ from another, but bursts from the same one will vary with the muscular effort exerted. Such inaccuracy is completely uncalled for in a situation involving the use of potentially dangerous drugs of which the total dose administered should be known.

In our clinic the test dose is administered by a nurse. With a 5-cc. syringe she withdraws the prescribed amount from a stock bottle and empties it into a small

16. Caution in Use of Butyn Sulfate and of Pontocaine, Letter from the Medical Director, Veterans Administration, July 6, 1942.

17. Veterans Administration Technical Bulletin, 10 A-106, Jan. 16, 1948.

18. Derbes, V., and Engelhardt, H.: Deaths Following the Use of Local Anesthetics in Transcricoid Therapy: Critical Evaluation, J. Lab. & Clin. Med. **29**:478, 1944.

solution glass. The solutions are weaker than those used later to anesthetize the tracheobronchial tree to afford greater volume for gargling. In the case of cocaine the test dose is 5 cc. of a 1% solution, or 50 mg. For tetracaine it is 4 cc. of a 0.25% solution, or 10 mg. The patient gargles for one-half minute with whichever of these anesthetics is to be used and expectorates. Five minutes or more later, intratracheal anesthesia is begun.

In our experience, if a patient tolerates this test dose without reaction, he is able to take the additional calculated amount needed for complete anesthesia. Although the particulars of technique may differ among endoscopists, certainly some preliminary test should be made in all cases, and the exact quantity of drug used should be known.

SPRAYS AND PYRIFORM SINUS APPLICATORS

The objection to sprays has already been expressed. They do not readily lend themselves to exact measurement of dosage, which is the most important single factor in preventing reactions. In those rare instances in which the total quantity in the spray bottle has been carefully measured out for each patient before use there can, of course, be no objection to atomizers.

Pyriform sinus applicators are open to the same criticism, but the likelihood of controlling them is somewhat better. If a calculated amount of anesthetic is measured into a solution cup for each patient and this total is never exceeded, it makes little difference how the applicators are used. Whether the applicator is dripping wet or wrung dry back into the cup, the total dose of anesthetic will be the calculated one. The danger lies in dipping the applicators into an unmeasured quantity of solution, wringing them out to varying degrees so that there cannot be the slightest knowledge of the number of milligrams of drug being delivered, and then following them up with further anesthesia by syringe. We have abandoned the use of these applicators because they have not offered anything beyond additional discomfort to the patient. For those who prefer them, there can be no objection if the quantity of drug involved in their use is known.

EPINEPHRINE

There is uncertainty concerning the role of epinephrine in topical anesthesia. This is partially attributable, as in other aspects of the problem, to drawing conclusions from laboratory experiments in which epinephrine was injected rather than applied topically. Braun [11] demonstrated in 1903 that epinephrine decreased the toxicity of injected local anesthetics. Fussgänger and Schaumann [10] found that the addition of epinephrine to tetracaine administered subcutaneously to rabbits had the same sparing effect. Gurd and Sachs,[19] however, in working with cocaine on white mice, demonstrated that epinephrine 1 : 85,000 was without effect on toxicity, but that concentrations of 1 : 50,000 or stronger increased toxicity. Ross [20] and Gold [21] found that epinephrine by intravenous injection increased the toxicity of cocaine in cats. These were all experiments by injection, and even these results are contradictory.

19. Gurd, M. R., and Sachs, I.: The Effect of Adrenalin on the Toxicity of Local Anesthetics, Quart. J. Pharm. & Pharmacol. **12**:713, 1931.

20. Ross, E.: Toxicity of Cocaine as Influenced by Rate of Absorption and Presence of Adrenalin, J. Lab. & Clin. Med. **8**:656, 1923.

21. Gold, H., cited by Mayer, E.: The Toxic Effects Following the Use of Local Anesthetics, J. A. M. A. **82**:876 (March 15) 1924.

One of us (H. J. R.[8]) investigated the effect of epinephrine on the toxicity of cocaine and tetracaine when administered topically to the tracheobronchial tree of rabbits. It was found that the period of time over which the test solutions were instilled was the most important element in determining the effect of epinephrine. For example, if a certain quantity of cocaine or tetracaine administered within 30 seconds invariably produced reactions of a particular severity, the addition of epinephrine with administration over the same period of time either enhanced the reaction or caused death. If, however, the solutions with epinephrine added were instilled more slowly, such as in four to five minutes, there was no perceptible change in reactions with cocaine and there was a decrease in toxicity with tetracaine. In other words, administration rapid enough to simulate intravenous injection by flooding some of the alveoli resulted in synergistic action between the anesthetic and epinephrine, with consequent increase in toxicity, thus bearing out the experimental findings of Ross and Gold. On the other hand, when administration was fractionated and sufficient time elapsed to allow epinephrine to exert its vasoconstricting effect (as in parenteral injection), there was a definitely favorable response in the case of tetracaine, minimum lethal doses being raised slightly less than one-third. There was no perceptible effect with cocaine 10% or 50%, but at least toxicity was not increased.

In the absence of clinical experimentation opinion must be based on the results of laboratory investigation. Certain conclusions may be drawn with regard to the effect of epinephrine on topical anesthetics in the tracheobronchial tree. It appears to afford some additional protection with tetracaine, none at all with cocaine. Those who use it should take care that administration is slow.

We do not use epinephrine, because it offers nothing when combined with cocaine and it so blanches the mucosa with tetracaine as to eliminate fine and even gross changes in color.

PERSONAL TECHNIQUE

Preliminary testing for sensitivity is conducted in the manner described under "Hypersensitivity." The gargle also mildly obtunds the gag reflex in most patients. When intrabronchial anesthesia is to begin, a nurse withdraws the prescribed amount of anesthetic solution with syringe and needle from the stock bottle and transfers it to a solution cup. The quantities employed are 8 cc. of tetracaine 0.5% (40 mg.) or 5 cc. of cocaine 5% (250 mg.). The reason for these particular amounts is discussed under "Total Dosage." The physician uses what is in the solution cup and no more. About 5 minutes after the preliminary gargle a small x-ray timer is set for 10 minutes, and intrabronchial anesthetization is begun. It is not completed before the bell rings. With a laryngeal mirror and a Lukens-Luer syringe the first few drops are directed to the epiglottis, then, after a minute or more, to the interior of the larynx, and finally to the tracheobronchial tree. The left bronchus is anesthetized by having the patient rest his left elbow on a pillow and then instilling fractionally the last half of the solution. After each fractional instillation the patient is encouraged to cough in order to facilitate distribution of the anesthetic throughout the area of instrumentation and to discourage descent into the smaller bronchi and alveoli where anesthesia serves no useful purpose. Total quantities of drug used in the procedure, including those in preliminary testing, are 300 mg. of cocaine or 50 mg. of tetracaine.

Though not by design, the dose of cocaine used by us was found to parallel closely that recommended by St. Clair Thomson[22] over 25 years ago. He advised a test dose of 1 grain

22. Thomson, St. C.: Diseases of the Nose and Throat, Ed. 3, New York, D. Appleton & Company, 1926, p. 77.

(60 mg.) to check for sensitivity; this was followed later by a solution containing not more than 5 grains (300 mg.).

CHOICE OF DRUG

Responses to questionnaires [23] have suggested that cocaine is in somewhat greater use than tetracaine, but each drug has its advocates. Efforts are made periodically to establish the superiority of one over the other, but nothing decisive has as yet come to light, and preference is a personal matter. Comparison of toxicity reports tells us only that individuals have died from both drugs, and we wonder how many reactions and deaths have occurred and not been reported.

The frequently voiced objection to tetracaine that it is more toxic than cocaine is without real significance. Milligram for milligram tetracaine is more toxic. In fact, it has been demonstrated experimentally [6] that it is six times as toxic in the tracheobronchial tree, and three times as toxic by subcutaneous injection as cocaine. However, the anesthetic potency of tetracaine is correspondingly greater, and a lesser amount is needed to achieve the same anesthetic effect. Since tetracaine is dispensed in no more concentrated a solution than 2%, as compared with cocaine, which can be obtained in any concentration, the onus of reaction from too much tetracaine must rest entirely with the operator, not with the drug. One might advance the analogous specious argument that pentobarbital sodium is far superior to morphine as a preoperative sedative because 3 grains (180 mg.) of it can be administered, whereas only ¼ grain (15 mg.) of morphine is safe.

In our clinics we use both cocaine and tetracaine, and find little to choose between them. They are equally satisfactory and are alternated every six months. On the basis of experimental work we have varied our techniques from time to time, and have finally arrived at the one now in use. This series represents 350 anesthetizations with cocaine and 400 with tetracaine. There have been no reactions. If it became necessary to make a choice between cocaine and tetracaine, we should be hard put to make a decision.

Objections to tetracaine are several. It has been suggested by Thomas and Fenton [12] that its toxicity is greater in allergic individuals, notably asthmatics. If this is corroborated by enough clinical reports, it will constitute a genuine deterrent to the use of tetracaine. There is also some question about its irritant qualities. Jackson and McReynolds [1] believe that tetracaine has a tendency to cause an irritation which may persist for several days. Ireland [23c] noted that the tracheal mucosa of guinea pigs showed swelling and desquamation following the topical application of tetracaine but not following cocaine.

On the other hand, tetracaine has certain advantages. First, it is cheaper. Second, in doses of comparable anesthetic potency the number of cubic centimeters of tetracaine which may be safely administered is greater (assuming the toxicity ratio of tetracaine to cocaine to be even as much as 6:1). The amounts of these anesthetics which may be instilled into the tracheobronchial tree are small as it is, and the additional quantity of tetracaine allows that much greater coverage of the tracheobronchial tree. Third, it has been demonstrated experimentally [6] that reactions to

23. (*a*) Jackson and McReynolds.[1] (*b*) Himalstein.[2] (*c*) Ireland, P. E.; Ferguson, J. K. W., and Stark, E. J.: The Clinical and Experimental Comparison of Cocaine and Pontocaine as Topical Anesthetics in Otolaryngological Practice, Laryngoscope **61**:767, 1951.

overdosage occur considerably more rapidly with tetracaine than with cocaine. In other words, although toxic quantities of both drugs are administered, manifestations of excessive dosage are several minutes slower to make their appearance in the case of cocaine. This is presumably due to the vasoconstricting action of cocaine, which slows its absorption. Slow onset of reaction, when one of the same severity is going to occur anyway, is a disadvantage. From a clinical standpoint, the administration of an excessive quantity of anesthetic may be associated with quicker evidence of reaction in the case of tetracaine. Thus, the operator may be able to withhold further anesthesia if using tetracaine, whereas additional cocaine might easily be given before a developing reaction became clinically evident. Where hypersensitivity rather than overdosage is responsible for reaction, this hypothesis is not valid.

At the time of writing, presumed advantages of either drug over the other are lost in the great success which both enjoy clinically. A strong stand in favor of one bespeaks a strictly personal rather than factual preference, and much more evidence must accrue before undisputed superiority of one can be established. As stated above, we use both drugs and consider them to be of equal clinical worth.

COMMENT

In summarizing the problem of topical anesthesia in bronchoscopy, it is important to differentiate between that which is basically important and that which is subordinate. The prime consideration in establishing a safe technique is controlled dosage. In this regard the difference in psychology between injection and topical anesthesia is difficult to reconcile. Cocaine and tetracaine are two to four times as toxic when applied experimentally to the tracheobronchial tree as when injected subcutaneously. Yet a physician who would not dream of injecting a drug without knowledge of the exact amount does not hesitate to call for "just one or two more sprays." The dosage in all patients should be calculated in milligrams and limited to that amount. Although we feel that the quantities of cocaine and tetracaine semi-officially held to be safe are too low to be practical and have raised them to what we believe are clinically effective levels, these amounts are carefully measured and never exceeded. Inadvertent overdosage, including such other blunders as using the wrong or a partially evaporated solution, can be attributed to nothing but carelessness. Although there may be differences of opinion among endoscopists as to optimal doses, there should be no disagreement on the necessity for careful measurement of the total dose administered and restriction to that amount.

The second important consideration is preliminary testing for hypersensitivity. All patients, including those who have received topical anesthesia in the past without incident, should be surveyed for decreased tolerance. Application of small measured amounts of anesthetic to the oral and pharyngeal mucous membranes is one satisfactory method.

The third basic consideration is control of the speed of administration. Slow fractional instillation and avoidance of large quick bursts keep blood levels from attaining sporadically high dangerous peaks.

These three salient elements—controlled total dosage, testing for hypersensitivity, and slow fractional administration—are the keystones of safe topical anesthesia in bronchoscopy. Other factors such as choice of drug, concentration, the use of

epinephrine, pyriform sinus applicators, etc., although important, relate more to refinements of technique and do not constitute the hub of the problem. In these matters there is greater opportunity for latitude, that is, variations in technique to suit the needs of the individual endoscopist. Since no official body has within recent years reviewed the question of topical anesthesia or made specific recommendations, each endoscopist must draw his own conclusions and make his own decisions. Close attention both to broad aspects of the problem and to details of technique should be rewarded by a decrease in the number and severity of reactions.

SUMMARY AND CONCLUSIONS

Topical anesthesia in bronchoscopy is reviewed in the light of recent experimental and clinical studies.

It is emphasized that the three basic considerations in establishing a safe technique are controlled total dosage, preliminary testing for sensitivity, and slow fractional instillation.

Currently accepted maximum safe doses of cocaine and tetracaine are too low and should be officially raised.

The entire problem of topical anesthesia should be taken under advisement by a national otolaryngologic society and, after appropriate studies, official recommendations should be made.

3875 Wilshire Blvd.

ALLERGY OF THE INNER EAR

(Ménière's Disease)

HENRY L. WILLIAMS, M.D.
ROCHESTER, MINN.

ALLERGY, whether it is of the immunologic or physical type, appears to be basically a localized autonomic dysfunction of the cholinergic type. This dysfunction produces a stereotyped reaction of the peripheral vascular bed (the arteriole, capillary, and venule) in which arteriolar spasm is associated with dilatation of the capillary and venule that sometimes amounts to a varicosity. Müller termed this condition the "spastic-atonic state." Anoxia of the capillary loop, with injury to the endothelium and contained circulating cells, follows. Such anoxia results in "sludging" of the blood, local release of toxic metabolites, and increased permeability of the capillary loop. The last, in turn, results in the formation of the typical allergic wheal.

When allergy affects the inner ear, this invariable reaction of the capillary bed may involve either the stria vascularis or branches of the internal auditory artery supplying the cristae and maculae, or the two together. Involvement of the stria vascularis should result in an increased volume of endolymph with an increased protein content, and consequently higher osmotic pressure. Involvement of the branches of the internal auditory artery supplying the cristae and maculae by producing local anoxia may give rise to crises of vertigo. Involvement of the two areas at once may give rise to the typical "triad" of Ménière's disease.

Although immunologic allergy may be the precipitating factor in Ménière's disease, physical allergy appears to be the more usual.

When the stria vascularis is involved, increased transudate with increased protein content goes into the endolymph system producing hydrops.

Endolymphatic hydrops is considered to be primarily a distention of the cochlear duct with secondary extension to the utricle and otic sac.

Dix, Hallpike, and Hood,[1] in discussing the pathologic changes in Ménière's disease, stated that characteristic alterations are to be seen in Corti's organ. The cell mass is compressed, the cell outlines are obscured, and the staining differentiation between nuclei and cytoplasm is virtually extinguished. These abnormalities are so striking, according to Cawthorne,[2] that it is inevitable that they should be regarded as distinctive of Ménière's disease in certain of its phases. That they are found

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From the Section of Otolaryngology and Rhinology, Mayo Clinic.

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1. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Observations upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve, Proc. Roy. Soc. Med. **41**:516-526 (Aug.) 1948.

2. Cawthorne, T.: Ménière's Disease, Ann. Otol. Rhin. & Laryng. **56**:18-38 (March) 1947.

sometimes and not always means only that they represent a transient and reversible reaction on the part of the hair cells to the chemicophysical disturbances which go with the gross distention of the endolymph system. It seemed reasonable to Cawthorne that this change should be phasic in character, thus matching the clinical course of the disease, and too, that the morphologic changes in the hair cells should vary at the same time.

Dix, Hallpike, and Hood [3] tended to correlate the compressive changes found in the organ of Corti with the deafness and particularly with the phenomenon of recruitment of loudness originally described by Fowler.

Because the endolymphatic membrane is a semipermeable membrane, and because ordinarily the osmotic pressures of the two humors are nearly the same, many authors have found it difficult to explain satisfactorily the pathologic finding of Hallpike and Cairns [3] that Reissner's membrane and the remainder of the endolymphatic membrane is ballooned out and the organ of Corti compressed.

A reasonable explanation appears to be that the semipermeable membrane of the endolymphatic system is relatively impermeable to protein molecules. Therefore the Donnan effect would be relatively large in such a situation, since it is directly proportional to the concentration of the nondialyzable substance. This would result in increased osmotic pressure in the endolymph system. The solvent water would then be drawn into the system from the perilymph, and distention of the membrane with increased pressure on the organ of Corti would result.

Arnvig [4] and Berggren [5] reported apparently authentic cases of Ménière's disease without hydrops in which no alterations could be found in the labyrinth. Brunner [6] reviewed the literature on the pathologic changes of Ménière's disease and stated that in some of his own cases in which the patients had suffered from recurring dizzy spells hydrolabyrinth was not found at necropsy. He cited Wittmaack to the effect that hydrolabyrinth may consist of distention of the membranous labyrinth owing to hypersecretion of endolymph. He stated that serous labyrinthitis or hydrops will not invariably cause hydrolabyrinth and that hydrolabyrinth may be a reversible process, although it eventually becomes irreversible. By hydrolabyrinth Brunner [6] apparently means an increase of fluid and protein material in both the endolymphatic and the perilymphatic spaces, while by serous labyrinthitis he appears to indicate an increase in fluid without a concomitant increase, or possibly even a reduction, in its protein content.

Brunner [6] stated that Ménière's disease was essentially vasomotor labyrinthitis. He argued that vasomotor labyrinthitis may or may not cause the symptoms of both vertigo and deafness. He stated that the histologic findings of an accumulation of albuminous fluid in the inner ear are not invariably associated with cochlear plus labyrinthine symptoms, regardless of whether the abnormal permeability of the blood vessels is inflammatory in origin or not. His investigations and reasoning left Brunner at a loss for an explanation of the facts he had observed. Much of his

3. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625-654 (Oct.) 1938.

4. Arnvig, J.: Histological Findings in a Case of Ménière's Disease, with Remarks on the Pathologic-Anatomical Basis of This Lesion, Acta oto-laryng. **35**:453-466, 1947.

5. Berggren, S.: Histological Findings in Ménière Cases, Acta oto-laryng. (Supp.) **78**:108, 1949.

6. Brunner, H.: Ménière's Disease, J. Laryng. & Otol. **62**:627-638 (Oct.) 1948.

difficulty in forming a reasonable hypothesis to explain the divorcement of the cochlear and vestibular symptoms in Ménière's disease seems to have been due to two factors: 1. His criteria for the diagnosis of Ménière's disease differ somewhat widely from those of Ménière and many other observers. 2. In considering the pathologic basis of Ménière's disease, he concentrated his observations exclusively on the postmortem histopathology and ignored the lessons of physiopathology which have long been pointed out by observers using the technic of biomicroscopy.

Békésy [7] cast doubt on the hypothesis presented by Dix, Hallpike, and Hood,[1] and Cawthorne [2] that compression of the cells of the organ of Corti produced by increased pressure in the endolymph would produce the characteristic changes of hearing found in endolymphatic hydrops. He was unable to show experimentally that increasing the pressure of the perilymph and endolymph produced a reduction in hearing. Lempert and his associates,[8] in confirming Békésy's work, stated that their observations showed, in addition, that increase of pressure in the humors did not interfere with the functioning of the sensory cells of the organ of Corti under the conditions of their experiments.

The evidence secured from the human being with endolymphatic hydrops appears to be directly opposed to that presented by Békésy from experiments on fresh cadavers and by Lempert and his associates [8] from experiments on the monkey.

Altmann and Waltner [9] called attention to the fact that the fluid exchange in the inner ear takes place through semipermeable membranes and the fluids involved are not simple, true solutions as in the experiments. The weight of evidence now favors the position that the conditions of the experiments done by Lempert and his associates do not reproduce the conditions in the inner ear in Ménière's disease, and that therefore their evidence may be misleading.[10]

In times past the deafness of Ménière's disease was referred to as being of the nerve type. Mygind [11] indicated that such statements were in error. He suggested that the deafness, while of an inner ear type, was in all probability due to changes in the "humors" of the inner ear and therefore was due to interference with conduction in the inner ear.

Wright [12] pointed out that the deafness of Ménière's disease may precede any other sign of the disorder by months or years. He termed this type of deafness "cochlear deafness" to distinguish it from the loss of hearing observed in degeneration of the fibers of the auditory nerve or spiral ganglion.

A characteristic feature of the deafness, at least in the early stages of Ménière's disease, is its tendency to increase and decrease in a seemingly capricious manner.

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7. Békésy, G.: Ueber der Schweigungen der Schneckentrennewand beim Präparat und Ohrenmodell, Akustische Ztschr. **7**:173-186, 1942.

8. Lempert, J.; Wever, E. G.; Lawrence, M., and Meltzer, P. E.: Perilymph: Its Relation to the Improvement of Hearing Which Follows Fenestration of the Vestibular Labyrinth in Clinical Otosclerosis, Arch. Otolaryng. **50**:377-387 (Oct.) 1949.

9. Altmann, F., and Waltner, J. G.: The Circulation of the Labyrinthine Fluids: Experimental Investigations in Rabbits, Ann. Otol. Rhin. & Laryng. **56**:684-708 (Sept) 1947.

10. Waltner, J. G., and Raymond, S.: On the Chemical Composition of the Human Perilymph and Endolymph, Laryngoscope **60**:912-918 (Sept.) 1950.

11. Mygind, S. H.: Experimental-Histological Studies on the Labyrinth: XI. Correlation of Our Histological and Clinical Experiments and Observations; Conclusive Review, Acta otolaryng. **35**:108-129, 1947.

12. Wright, A. J.: Menière's Disease, Proc. Roy. Soc. Med. **41**:801-805 (Nov.) 1948.

As shown by Lillie, Horton, and Thornell,[13] ability to hear may vary within a range of from 10 to 30 db. in the course of several days. These changes are not abrupt, however, and the fluctuations in hearing may be entirely independent of the presence or absence of vertigo. Dederding [14] showed that this "cochlear" deafness of patients who have Ménière's disease can be influenced by the intake of salt and fluid. Her evidence indicated also, she stated, that increasing the intake of sodium and fluid could precipitate the vertiginous crisis of Ménière's disease. Furstenberg, Richardson, and Lathrop [15] extended the investigations of Dederding and demonstrated that increasing the intake of sodium alone would precipitate a vertiginous crisis of Ménière's disease, whereas increasing the intake of fluid and restricting the intake of sodium would not precipitate one. They concluded that it was the excess of sodium itself, not "water loading" secondary to the ingestion of sodium, as Dederding stated, that precipitated the vertiginous crisis in this experiment.

The evidence furnished by Stoesser and Cook [16] and Kern [17] indicates that an excessive intake of sodium lowers the threshold for allergic reaction. It seems possible that the precipitation of the vertiginous crises of Ménière's disease in these so-called water-loading experiments, which was presumed to have been due to increased pressure in the endolymph, may have in fact been precipitated by an excess of the sodium ion, producing vasospasm in the branches of the internal auditory artery.

More than 40 years ago Shambaugh [18] suggested the hypothesis that the recurring vertiginous crises of Ménière's disease were produced by vasospasm. He pointed out that, owing to the anatomic arrangement disturbance of the circulation to the labyrinth may be limited to one or several of the terminal arterioles supplying certain areas without involving the remainder of the labyrinth.

Lermoyez [19] elaborated the hypothesis that circulatory dysfunction was the precipitating cause of the crisis. He suggested that deafness, tinnitus, and vertigo might result from either dilatation or constriction of the vessels supplying the labyrinth. He pointed out that a spasm of the internal auditory artery would affect simultaneously the equilibrial labyrinth and the cochlea, which would manifest their trouble by the complete symptomatic triad of Ménière. Spasm of the branches to the vestibule, on the other hand, might precipitate paroxysmal vertigo without concomitant effect on the hearing, whereas spasm of the cochlear branches might

13. Lillie, H. I.; Horton, B. T., and Thornell, W. C.: Ménière's Symptom Complex: Observations on the Hearing of Patients Treated with Histamine, Ann. Otol. Rhin. & Laryng. **53**:717-741 (Dec.) 1944.

14. Dederding, D.: Our Menière Treatment (Principles and Results), Acta oto-laryng. **16**:404-415, 1931.

15. Furstenberg, A. C.; Richardson, G., and Lathrop, F. D.: Ménière's Disease: Addenda to Medical Therapy, Arch. Otolaryng. **34**:1083-1092 (Dec.) 1941.

16. Stoesser, A. V., and Cook, M. M.: Possible Relation Between Electrolyte Balance and Bronchial Asthma, Am. J. Dis. Child. **60**:1252-1268 (Dec.) 1940.

17. Kern, R. A.: The Role of Water Balance in the Clinical Manifestations of Allergy, Am. J. M. Sc. **199**:778-789 (June) 1940.

18. Shambaugh, G. E.: Some Relations in the Blood-Supply of the Inner Ear Which Have a Practical Bearing on the Clinical Study of Otology, Arch. Otol. **35**:11-19, 1906.

19. Lermoyez, M.: Le vertigue qui fait entendre: Angiospasme labyrinthique, Ann. d. mal de l'oreille, du larynx **48**:575-583 (June) 1929.

provoke deafness and tinnitus but no vertigo. Since Lermoyez' time most students of Ménière's disease have considered it a vasomotor labyrinthitis or, as it was recently put by Hilger,[20] vasomotor labyrinthine ischemia.

If the cochlear changes in Ménière's disease are considered due to the stereotyped vascular change of allergic autonomic dysfunction taking place in the stria vascularis with a secondary increased transudation of the proteins of the serum while the vertigo is on the basis of the same type of dysfunction of the peripheral vascular bed affecting the blood supply to the cristae and maculae and producing immediate stimulation by anoxia, a separation of phenomena which is definitely possible on an anatomic basis, there appears no particular reason why endolymphatic hydrops both with and without vertigo should not occur. Ménière's disease, also on the same basis, can be conceived of as occurring with and without endolymphatic hydrops.

The problem of allergy of the inner ear may be made more easily comprehensible by a brief review of the humoral systems and the blood and autonomic nerve supply to the labyrinth.

ANATOMIC AND PHYSIOLOGIC FACTORS

The Humoral System of the Labyrinth.—The humoral system of the labyrinth is made up of a completely closed system, the endolymph system, surrounded by a second system, the perilymph system, which in laboratory animals communicates with the arachnoid sac through the aquaeductus cochleae (perilymphatic duct). These two systems are separated for the most part by a semipermeable, flat endothelial membrane except where separation is produced by the basilar membrane. In man, under normal conditions, according to Aldred, Hallpike, and Ledoux,[21] the osmotic pressure of the endolymph is slightly greater than that of the perilymph.

Altmann and Fowler [22] stated that, although many authors assume that pressure equilibrium between the cerebrospinal fluid and the perilymph is maintained by the cochlear aqueduct in human beings as well as in animals, this is not necessarily so. They pointed out that Meuerman found this aqueduct in man to be relatively much narrower than in experimental animals and ordinarily filled with dense connective tissue and often with bone. Zange [23] was struck with the narrowness of the cochlear aqueduct and compared it to a curved capillary tube. He found that the viscosity of the perilymph and the cerebrospinal fluid was high and concluded that this would prevent sudden sharp rises in endolymphatic and perilymphatic pressure from being quickly transmitted to the subarachnoid space, even if the cochlear aqueduct were open. Waltner's [24] findings suggested that in the rabbit

20. Hilger, J. A.: Autonomic Dysfunction in the Inner Ear, Laryngoscope **59**:1-11 (Jan.) 1949.

21. Aldred, P.; Hallpike, C. S., and Ledoux, A.: Observations on the Osmotic Pressure of the Endolymph, J. Physiol. **98**:446-453 (Sept.) 1940.

22. Altmann, F., and Fowler, E. P., Jr.: Histological Findings in Ménière's Symptom Complex, Ann. Otol. Rhin. & Laryng. **52**:52-80 (March) 1943.

23. Zange, J.: Pathologische Anatomie und Physiologie der mittelohrentspringenden Labyrinthentzündungen als Grundlage der Klinik: Zugleich eine kurze Klinik dieser Erkrankungen, Wiesbaden, J. F. Bergmann, 1919.

24. Waltner, J. G.: Barrier Membrane of the Cochlear Aqueduct: Histologic Studies on the Patency of the Cochlear Aqueduct, Arch. Otolaryng. **47**:656-669 (May) 1948.

there is an endothelial membrane at the junction of the vestibule and the cochlear aqueduct. In his opinion, this showed that even in the rabbit an increase in volume and pressure of viscous fluids, such as the humors of the inner ear, probably would not be relieved by "free communication" with the arachnoid sac. Such communication might be obtained only in exceptional circumstances with rupture of the occluding membrane of the cochlear duct.

The recent work of Bast and Anson [25] appears to reassert the hypothesis that most of the endolymph is carried off through the otic sac. Most observers are of the opinion that perilymph originates from endolymph by transudation through the endolymphatic membrane. The osmotic pressures of the humors of the inner ear have been found to be definitely higher than the osmotic pressure of the cerebrospinal fluid. The slight difference in osmotic pressure between the endolymph and perilymph may be due to the Donnan effect, since the endolymph may be presumed to have a higher concentration of protein because of the possibility for the increased transudation of protein particles into the endolymph through the stria vascularis.

The organ of Corti receives all its nutrition from the endolymph, since no blood vessels supply it. For this reason it differs from other organs that receive vascular supplies. This difference, however, is more apparent than real, because all cells receive their nutrition from lymph. Anatomists have recognized that the humoral systems of the labyrinth are analogous and comparable to an enlarged lymph space in their nomenclature "perilymph" and "endolymph." The difference between the humoral systems and other lymph spaces lies in the greater size of the former. This factor, however, may be expected to have definite effects on the rapidity with which osmotic and chemical adjustments take place in the humors.

Since the work of Guild [26] the stria vascularis has been generally accepted as the source of endolymph. Shambaugh,[27] however, described certain glands in the sulcus externus which he considered to be a partial source of the endolymph. Wittmaack [28] stated that the endolymph was formed from a transudate from the vessels of the stria vascularis and a secretion from glands, the former rich in protein and the latter similar to a saline solution. Wakim [29] considered this a reasonable assumption because physiologists are beginning to consider that all serous fluids of the body are a combination of a transudate from the capillaries and of glandular secretion. This combination would offer a mechanism by which compensation for changes in the osmotic equilibrium could be rapidly effected.

Guild [26] found that the endolymph in the cochlear duct moves toward the basal end and passes through the ductus reuniens into the otic sac (endolymphatic sac). He concluded that the endolymph passes through the wall of that part of the otic

25. Bast, T. H., and Anson, B. J.: Postnatal Growth and Adult Structure of the Otic (Endolymphatic) Sac, Ann. Otol. Rhin. & Laryng. **59**:1088-1101 (Dec.) 1950.

26. Guild, S. R.: The Circulation of the Endolymph, Am. J. Anat. **39**:57-81 (Mar.) 1927.

27. Shambaugh, G. E.: On the Structure and Function of the Epithelium in the Sulcus Spiralis Externus, Arch. Otol. **37**:538-546, 1908.

28. Wittmaack, K.: Hydrops labyrinthi (Labyrinthitis serosa), In Die entzündlichen Erkrankungsprozesse des Gehörorganes, in Henke, F. and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Springer-Verlag, 1926, vol. 12, pp. 290-307.

29. Wakim, K. G.: Personal communication to the author.

sac which he termed the "pars intermedia." In his opinion structural differentiation indicated that this is a functionally specialized region. This opinion has recently been substantiated by Bast and Anson [25] and by Arnvig.[4] Bast and Anson answered some of the objections proposed by M'Nally [30] and Lindsay [31] to Guild's [26] hypothesis.

Saxén [32] and Kobrak [33] suggested that the endolymph was secreted by the cells of the stria vascularis and resorbed by the cells of the sulcus externus. Ruëdi [34] made some observations which he thought pointed to this conclusion. The weight of the evidence, however, appears to favor the hypothesis originally presented by Guild.[26]

In the opinion of Altmann and Waltner,[9] the fact that the endolymph and perilymph are practically iso-osmotic favors the assumption that the endolymph gives origin to at least part of the perilymph. They also stated that morphologic evidence suggested that the modiolar vessels might be a possible source of origin of part of the perilymph, especially in man and monkey.

The Elimination of the Labyrinthine Humors.—As mentioned before, the endolymph and perilymph systems appear to be analogous to lymph spaces in other portions of the body. Elsewhere in the body interstitial fluid tends to be eliminated via the lymph vessels. Movement of lymph generally is promoted by the contraction and relaxation of muscles and by vascular pulsation. In the inner ear, however, nature appears to have eliminated the possibility that such mechanical factors will aid in the elimination of interstitial fluid.

Pulsations of the internal carotid artery are largely eliminated when it passes through the carotid canal, as is indicated by the fact that under normal conditions the arteries of the retina do not pulsate. Thus reduction of all pulsation occurs within the cranium. The long narrow labyrinthine artery (or the anteroinferior cerebellar artery, whichever nomenclature is accepted) tends to diminish arterial pulsation in the inner ear. In addition, Mygind has described certain "glomeruli" in the arterial loops to the cochlea which tend to eliminate pulsations and other changes in pressure. Pulsations in the brain are prevented from affecting the humors of the inner ear by the long, narrow vestibular aqueduct designed physically to absorb and dampen any waves of alternating pressure transmitted from the brain and cerebrospinal fluid. Pulsations in the cerebrospinal fluid also are prevented from affecting the humors by the fact that in man the cochlear aqueduct (perilymphatic duct) does not appear to communicate with the arachnoid sac.

Therefore actual secretion, as suggested by Bast and Anson, and the hydrostatic and colloidal osmotic pressures, as suggested by Altmann and Waltner,[9] seem to be the factors that must be largely effective in the elimination of an increased endo-

30. M'Nally, W. J.: Experiments on the Saccus Endolymphaticus in the Rabbit, J. Laryng. & Otol. **41**:349-360 (June) 1926.

31. Lindsay, J. R.: Effect of Obliteration of the Endolymphatic Sac and Duct in the Monkey, Arch. Otolaryng. **45**:1-13 (Jan.) 1947.

32. Saxén, A.: Histologische Untersuchungen über die Sekretion und Resorption der Endolymphe im Innenohr, Bull. schweiz. Akad. med. Wissensch. **3**:250-261, 1948.

33. Kobrak, F.: Labyrinthliquor: Theoretische und angewandte Physiologie des Labyrinthliquors, Arch. f. Ohren-, Nasen-u. Kehlkopfh. **156**:30-112, 1949.

34. Ruëdi, L.: Some Experimental Findings on the Functions of the Inner Ear, unpublished data.

lymph of higher osmotic pressure, such as may be presumed to be present in Ménière's disease with endolymphatic hydrops.

Autonomic Nerve Supply to the Labyrinth.—The weight of the evidence favors the opinion that the adrenergic (sympathetic, thoracolumbar) nerve supply to the labyrinth is along the vertebral, basilar, and labyrinthine arteries.

Rasmussen [35] suggested that the cholinergic (parasympathetic, craniosacral) nerve fibers to the labyrinth originate in the superior olive and travel by the olivocochlear fasciculus to the vestibular branch of the eighth cranial nerve, reaching the cochlea via Oort's vestibulocochlear anastomosis. It has not been conclusively settled whether vasoconstriction in the labyrinth is produced by craniosacral or by thoracolumbar impulses. Experimental evidence seems to favor the latter conclusion slightly, but from the clinical standpoint the former seems the more reasonable.

The Vascular Supply to the Labyrinth.—The arterial supply to the labyrinth appears to be from a long narrow vessel termed by Siebenmann [36] the "internal auditory artery" and by Nabeya [37] the "antero-inferior cerebellar artery," which divides into the vestibular artery, the vestibulocochlear artery, and the cochlear propria artery.

Comment.—Even from this superficial résumé of the anatomy and physiology of the humoral system it can be readily concluded that several functional and pathologic changes may effect an increase in the fluid pressure of the endolymph and perilymph. To produce changes in the humors, the secretory mechanism, the absorbing mechanism, or the osmotic pressure relationships would have to be involved.

The relative slowness with which changes in the cochlear signs take place in Ménière's disease as opposed to the sudden onset and departure of the signs of disturbance of the equilibrial labyrinth indicates some difference in the mechanism producing these two different symptom complexes. Vasospasm producing anoxia, or sudden release of vasospasm producing irritation by a sudden resumption of blood flow, seems to explain the disturbances of the equilibrial labyrinth. In the cochlea, however, while the disorder may be initiated by changes in the stria vascularis, the relatively slow compensatory changes in the humors, as reflected by changes in hearing, must result from the slower changes in osmotic equilibrium initiated by changes in the stria vascularis and their effect on the composition of the endolymph. The evidence available points to involvement of the peripheral vascular bed in infection, chronic degenerative states, thrombosis, embolism, and allergy as the usual causes of hydrops involving the endolymph. In all these disorders except that produced by the localized autonomic dysfunction of allergy the resulting hydrops appears to be irreversible.

Factors such as "water loading," increased sodium intake, and the like may produce hydrops in a predisposed labyrinth, by which is meant a labyrinth already involved in an allergic mechanism.

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35. Rasmussen, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex, J. Comp. Neurol. **84**:141-219 (April) 1946.

36. Siebenmann, F.: Die Blutgefässe im Labyrinthe des menschlichen Ohres, nach eigenen Untersuchungen an Celloidin-Korrosionen und an Schnitten, Wiesbaden, J. F. Bergmann, 1894, 33 pp.

37. Nabeya, D.: A Study in the Comparative Anatomy of the Blood-Vascular System of the Internal Ear in Mammalia and in Homo (Japanese), Kyoto, Japan College of Medicine **6**:1-132, 1923.

THE DIFFERENTIAL DIAGNOSIS OF MÉNIÈRE'S DISEASE AND ENDOLYMPHATIC HYDROPS

Good therapeutic results in the allergies of the inner ear depend largely on their careful distinction from conditions which may simulate allergies and yet cannot be reversed by the therapeutic methods effective in the allergies.

Several cases illustrating some of the difficulties and pitfalls of differential diagnosis will be given.

Although the usual description of endolymphatic hydrops on an allergic basis suggests that differentiation from other types of deafness should prove comparatively easy, in my own experience this has not always proved to be so. Deafness due to endolymphatic hydrops is frequently described as perceptive in type and as readily separable from deafness due to otosclerosis or from other conduction types of deafness by the Rinne test. Results of this test are said to be always positive in perception deafness and negative in conduction deafness.

Although this usually holds, exceptions are occasionally met with. Dix, Hallpike, and Hood [1] pointed out that occasionally in the deafness of Ménière's disease the Rinne test gave a negative result even when absolute bone conduction was tested for. This has been my experience also. I believe it is due to two factors: (1) patients who are completely unable to differentiate sound and vibration, and (2) an even smaller group of patients in whom a negative result of the Rinne test is actually obtained in endolymphatic hydrops. Dix and Hallpike [38] suggested that the negative result in the latter group is due to a considerable increase in the density of the labyrinthine fluid if not to the formation of an actual coagulum. When a negative Rinne test is associated with a history of slowly progressive deafness over many years accentuated by pregnancy, the differentiation from otosclerosis is not at once obvious. This type of case, however, is usually not allergic in origin and, therefore, probably should not be classified as Ménière's disease. The following patient presented this difficulty in diagnosis:

CASE 1.—A white woman, 36 years of age, stated that she had first noticed deafness at the age of 13 years. There was not much further change in hearing until she noticed a considerable progress in her deafness with the birth of each of her two children. She had never had any tinnitus, vertigo, or nausea. She felt that she heard better in a noisy place than in a quiet one but felt she heard women's voices better than men's. She had worn a hearing aid for more than five years but did not think that it improved her ability to hear in the slightest degree.

The Rinne test with tuning forks gave negative results in both ears at 256 cps. This patient had been referred to me for consideration for the fenestration operation. Results of general and laboratory examinations were essentially negative except for the hearing tests. Threshold audiograms for pure tones and discrimination test for phonetically balanced words were obtained (Chart 1).

These findings seemed to indicate endolymphatic hydrops. While a loudness balance test could not be done, it was felt that the minimal interval between the speech reception threshold and the level of most comfortable loudness in both ears as well as the discrimination deafness indicated the presence of endolymphatic hydrops.

It might be argued that endolymphatic hydrops of allergic type had occurred secondarily in two ears already the seat of otosclerosis, but nothing in the history

38. Dix, M. R., and Hallpike, C. S.: Observations on the Pathological Mechanism of Conductive Deafness in Certain Cases of Neuroma of the VIII Nerve, Proc. Roy. Soc. Med. **43**:291-298 (April) 1950.

points to the onset of such a condition. This patient had gone 23 years since the onset of her deafness without either vertigo or dizziness. I subscribed to the hypothesis that the loss of hearing was on the basis of an endolymphatic hydrops from the first, especially since I had seen several somewhat similar cases. The essentially vascular histopathologic change shown by Wolff [39] to be present in otosclerosis seems to make the association of otosclerosis with endolymphatic hydrops a defensible suggestion, however. This type of endolymphatic hydrops has been found to be completely irreversible under treatment; hence, the fundamental physiologic mechanisms of its production may be different from those of the reversible variety. I have also found, however, that severe deterioration of discrimination for phonetically balanced words may not always be present in what appears to be a definite instance of endolymphatic hydrops not on an allergic basis. The following case appears to illustrate this point.

Case 2.—A man, aged 43 years and weighing 252 lb. (114.3 kg.), who had reduced his weight from more than 350 lb. (158.7 kg.) in the previous year, stated that he had had one

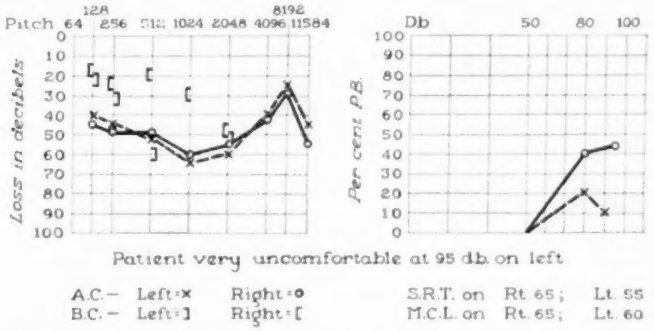
Pitch 64 128 256 512 1024 2048 4096 8192 11584
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
Db 50 80 100
Per cent P.B.
100 90 80 70 60 50 40 30 20 10 0
Patient very uncomfortable at 95 db on left
A.C.— Left=× Right=○
B.C.— Left=] Right=[
S.R.T. on Rt. 65; Lt. 55
M.C.L. on Rt. 65; Lt. 60

Chart 1 (Case 1).—These audiometric findings appear to indicate the presence of endolymphatic hydrops. The patient, a woman 36 years of age, was referred for a fenestration operation because of negative results of the Rinne test.

severe attack of vertigo, nausea, and vomiting in each of the years 1937, 1938, 1939, 1945, and 1949. In the spring of 1950 (11 months before being seen at the Mayo Clinic) he had 10 severe attacks in 10 days. At that time severe tinnitus, which was somewhat more marked on the left, developed and hearing diminished in both ears. Since that time two weeks had been the longest period he had passed through without severe attacks of vertigo, nausea, and vomiting. At the start of the severe attacks he had been given benzazoline (priscoline®), which seemed to give him partial relief for a time. He then was given nicotinic acid in doses up to 500 mg. three times a day. This produced little flushing and no symptomatic relief. He was then given a diet containing 0.2 gm. of sodium or less, and was given daily intravenous injections of 0.2% solution of procaine hydrochloride with epinephrine and ascorbic acid for two weeks, without effect. Next Jacobson's solution (benzyl cinnamate) and then six injections of corticotropin (ACTH) were tried without effect. Finally dimenhydrinate (dramamine®), 50 mg. every three hours, together with ¾ grain (0.05 gm.) of phenobarbital every three hours, was tried. He obtained more marked relief from this treatment than from any of the other medicaments; the attacks became rather mild and lasted 1 to 10 minutes. They continued to be frequent, however, and he was completely incapacitated for his usual occupations.

39. Wolff, D.: Otosclerosis: Hypothesis of Its Origin and Progress, Tr. Am. Acad. Ophth. (Supp.), pp. 11-22 (Oct. 8) 1949.

General physical examination was without particular significance except for the finding of glycosuria, Grade 3, on a grading basis of 1 to 4, and 195 mg. of sugar per 100 cc. of blood. The diabetes was not severe, and it was thought that it would become milder with further loss of weight. A 1,000 calorie diet was recommended, and he was advised to reduce his weight to 175 lb. (79.4 kg.). With this diet and without insulin his urine became sugar-free.

The results of an examination of the central nervous system, which included roentgenograms of the head with Stenver's projection to show the internal auditory meatuses, were negative.

Examination of the ears, nose, and throat gave negative results except for the audiometric findings (Chart 2 and Table). The speech reception threshold in the right ear was 25 db.,

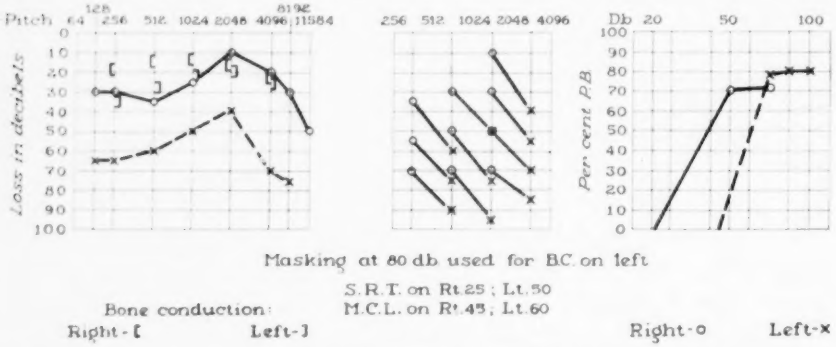

Chart 2 (Case 2).—Probable diabetic vasculitis with bilateral endolymphatic hydrops. Endolymphatic hydrops is indicated by the narrow "dynamic range" in the two ears (the difference between the speech reception threshold and the minimal comfort level). The lack of evidence of recruitment shown on the loudness balance test is thought to be owing to the presence of hydrops on both sides. The lack of discrimination deafness present seems to indicate an absence of compression of the organ of Corti.

Equilibration Test *

Canals Stimulated	Ice Water, Cc.	Nystagmus: Type	Degree	Amplitude	Duration	Vertigo
Right horizontal	5	→	2	2	55 sec.	Mild
	10	→	2	2	1 min.	
	30	→	2	2	1 min. 25 sec.	
Left horizontal	5	←	3	2	1 min. 40 sec.	Moderate

* No spontaneous nystagmus or positional nystagmus; hypoactive labyrinthine response on left.

and in the left 50 db. The level of most comfortable loudness in the right ear was 45 db., in the left 60 db.

In spite of the somewhat equivocal audiometric findings, a diagnosis of Ménière's disease with endolymphatic hydrops was made. The patient was given histamine intravenously without any observable effect. The diagnosis was changed later to endolymphatic hydrops on the basis of diabetic vasculitis.

The lack of recruitment in the loudness balance test in Case 2 may be misleading. In my experience, if hydrops is present on both sides, recruitment may not be obtainable. This is borne out by the narrow interval found between the speech reception threshold and the most comfortable loudness for speech on both sides. In spite of this, however, the discrimination curve for phonetically balanced words rises to 65 and 70% on both sides.

It may appear that I was in error in classifying this as a case of Ménière's disease. It is well known that vascular changes are common in diabetes and that angina and claudication are not infrequently present. It is probable that the dysfunction of the peripheral vascular bed here depended on diabetic changes in the endothelium rather than on autonomic imbalance. Certainly drugs directed toward correcting autonomic imbalance proved ineffective. Dimenhydrinate and phenobarbital, which appear to have a similar sedative action on higher centers, gave some measure of relief.

CASE 3.—The patient, a musician aged 27 years, stated that she had had a hearing loss in the left ear since she had measles in childhood. She felt that there was a difference of about a quarter tone in the perception of a given pure tone as heard in the two ears. She had never noted any variation in her hearing and had never experienced vertigo, dizziness, or unsteadiness.

Ear, nose, and throat examination gave negative results except for the hearing tests. Diplacusis was evident in the right ear as compared with the left in the following frequencies: higher at 128 cps, equal at 256, and lower at 512, 1024, and 2048.

A pure tone threshold audiogram showed normal hearing in the right ear. On the left findings in decibels for air-conducted sounds at various cycles per second were as follows:

128	256	512	1024	2048	4096
30	30	25	30	40	35

Bone conduction was close to the 30 db. level at all frequencies in the left ear.

Loudness balance tests showed marked recruitment of the left ear over the right. Hyperrecruitment was present at frequencies of 512 and 1024 cps, and recruitment was complete at the frequency of 2048 cps.

It was felt that the hearing loss in this ear in all probability represented a permanent irreversible hydrolabyrinth resulting from an acute labyrinthitis associated with measles in childhood.

CASE 4.—The patient, a man 38 years of age, presented himself to us because of attacks of vertigo with occasional nausea and vomiting and increasing deafness which had first appeared two years previously. The first attack began with dullness in the right ear while the patient was driving his car. Two hours later his surroundings suddenly appeared to be spinning in a counterclockwise direction. He stopped his car, got out, and tried to walk, but could not keep his balance. The attack lasted about two hours. He had six somewhat similar attacks in the succeeding two years, with vomiting on three occasions. During the attacks hearing in the right ear decreased but returned to normal after the attacks until four months before his admission, when he began to notice intermittent tinnitus with gradually decreasing hearing on the right.

During the year before admission he took ammonium chloride tablets when he felt an attack coming on, with, he believed, some symptomatic relief. He had also had a submucous resection performed a year before his admission, but with no relief to his symptoms. Results of the ear, nose, and throat examination were negative except for the findings relative to the functions of the inner ear.

The threshold audiogram for pure tones showed normal findings on the left. On the right the findings in decibels at various frequencies were as follows:

	128	256	512	1024	2048	4096
A. C.	65	60	65	65	50	20
B. C.	0	60	45	30	35	..

Loudness balance tests showed an incomplete recruitment of loudness in the right ear over the left at frequencies of 512, 1024, and 2048 cps.

Testing of the equilibrial function of the labyrinths by a modification of the Kobrak test showed relatively decreased equilibrial function on the right. Examination of the eyes revealed

normal pupils, reflexes, and fundi. A roentgenogram of the skull, however, showed evidence of Paget's disease with osteoporosis circumscripta especially involving the left frontoparietal region, and platybasia.

It has been recognized for a long time that Paget's disease of the skull may be associated with hydrops and recurring attacks of vertigo that are not reversible under treatment. Nevertheless, this patient was given our standard treatment for Ménière's disease with labyrinthine hydrops in the forlorn hope that in this instance the hydrops might be an intercurrent disorder. However, after a month's trial no benefit was secured. The patient was then given dimenhydrinate, 50 mg. three times a day, which also failed to give relief.

CASE 5.—Ten years before his admission to the clinic the patient, a man aged 61 years, noticed while driving his car that the road seemed to be coming up into his face. He stopped the car, got out, and vomited. After getting back in he was unable to drive. He was unable to walk for three days and had to be led to and from work for the next three months. He has never been entirely free from a sensation of unsteadiness and dizziness, but this symptom waxed and waned and was likely to be more pronounced on rising in the morning.

A month after the onset of his complaint he noticed a throbbing in his right ear synchronous with his pulse. Excitement or effort tended to make this throbbing more pronounced.

Ménière's syndrome was diagnosed elsewhere, but treatment had not given any symptomatic relief. Treatment consisted of low-salt diet, intravenous injections of histamine, and tubal insufflations. He had little disturbance in hearing, but at times he could not hear a watch tick (he was a watchmaker) and at other times he heard a watch tick better in one ear than in the other. He felt that his symptoms were distinctly less pronounced at the time of his admission than they had been 10 years previously.

Results of ear, nose, and throat examinations were essentially negative, except for the findings in regard to the function of the inner ear.

Audiometric tests with pure tones at threshold gave the following findings in decibels:

	128	256	512	1024	2048	4096
A. C. Rt.	15	15	15	10	25	40
A. C. Lt.	5	0	0	0	10	10
B. C. Rt.	..	15	15	15	25	..
B. C. Lt.	..	0	50	35	40	..

Recruitment was not present. The patient had a positional vertigo without nystagmus on changing from the supine to the sitting position. Tests of function of the equilibrial labyrinth by a modification of the Kobrak test with 5 cc. of ice water produced nystagmus of normal duration, direction, and amplitude on both sides, but vertigo could not be elicited even when the amount of the ice water used was increased to 30 cc. In other words, there appeared to be a dissociation of vertigo and nystagmus which, according to Leidler, is produced by a lesion in the substantia reticularis.

On examination of the eyes the pupils and reflexes were found to be normal, and the fundus and fields negative. Roentgenograms of the skull, including the Stenver and Towne projections, showed nothing abnormal, and general physical examination gave negative results except for questionable mild anemia. Except for the ear findings, examination of the central nervous system gave objectively normal results. It was felt that the diagnosis of a non-neoplastic lesion of the brain stem, possibly a small thrombus with infarction, would fit in well with the history and findings. The patient was given dimenhydrinate, 50 mg. three times a day, which produced no effect on his symptoms.

Although it is true that the symptoms and signs in this case are not much like those of Ménière's disease, the patient nevertheless was treated for "Ménière's syndrome" by several physicians. This should serve as a warning to us not to confuse ourselves by resorting to this meaningless diagnostic term but rather to attempt differential diagnosis.

In studying a group of approximately 500 cases of Ménière's disease with endolymphatic hydrops, I felt that the finding of vestibular hypofunction in the affected ear was a sign of significance. This was particularly true in cases of cochlear deafness. However, unilateral vestibular hypofunction is so frequently found in angle lesions that testing the equilibrial mechanism is not of marked value in the differential diagnosis.

Onset of cochlear deafness is usually slow and insidious. Nevertheless, Williams, Horton, and Day [40] found instances of precipitous onset and recovery and other instances in which sudden onset was not followed by remission. At that time they classified the latter as instances of thrombosis or embolism. In the next case, sudden onset of deafness was associated with recruitment and yet vigorous therapy failed to produce any change.

CASE 6.—This patient, a white woman aged 23 years, had noticed slight deafness in the left ear during the winter five years before her present admission. This had appeared with no obvious cause and disappeared the following summer. She had noticed no further trouble until six weeks before coming to the clinic, when she suddenly found she could not hear in her left ear. The patient was a school teacher in the primary grades, so it may be presumed that the loss of hearing appeared as suddenly as she assumed.

A general physical examination, including an examination of the fundus, a roentgenogram of the head with Stenver's projection to show the internal auditory meatuses, and a Wassermann test for syphilis, gave negative results. Tests of hearing disclosed marked diplacusis binauralis dysharmonica at 128 and 256 cps, and slight diplacusis at 512 cps. No diplacusis could be made out at 1024 cps. The tuning forks were heard as of higher pitch in the left ear.

Caloric testing of the function of the equilibrial labyrinth by a modification of the Kobrak method gave normal findings on both sides. The speech reception threshold on the right was 0 db., on the left 50 db.; the most comfortable loudness of speech on the right was 45 db. and on the left 70 db. In the right ear 98% of phonetically balanced words were received at 45 and 60 db. On the left 72% of the words were received at 70 db., and 60% were received at 85 db. Hearing of pure tones in the right ear was normal, by both bone and air conduction. Decibel levels at which various pure tones were heard in the left ear by air conduction are as follows:

128	256	512	1024	2048	4096	8192	11584
30	40	45	50	45	45	65	65

With masking at 70 db. bone conduction was found in the left ear at 40 db. for frequencies of 256 cps, and at 60 db. for 2048 cps only.

Loudness balance tests showed slight partial recruitment at frequencies of 500 and 1000 cps. At 2000 cps slight recruitment was present until 70 db. was reached, when complete recruitment suddenly appeared. At 80 db., however, there was again a 15 db. difference between the two ears. This finding was rechecked several times and has proved difficult to explain.

In spite of these somewhat equivocal findings, the patient was given treatment for a presumed endolymphatic hydrops for two months but failed to show any change in symptoms or signs.

The only conditions in which it is suggested that hydrops may be produced by interference with absorption of the humors are the early stages of neuromas of the eighth nerve and angle tumors. In these conditions choking of the labyrinth has been said to originate from interference with the venous outflow.

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40. Williams, H. L.; Horton, B. T., and Day, L. A.: Endolymphatic Hydrops Without Vertigo; Its Differential Diagnosis and Treatment, Tr. Am. Otol. Soc. **35**:116-142 (April) 1947.

Cases of this type are rarely recognized early enough so that the findings of recruitment suggesting a labyrinthine hydrops predominate over the signs produced by degeneration of the nerve cells and fibers of the eighth nerve. However, Case 7 well illustrates the phenomenon of recruitment in an angle lesion.

CASE 7.—A man aged 38 years stated that four years before admission to the clinic he became aware of drawing movements of the right side of the face, worm-like in speed and behavior. They were most marked in the chin, which became deeply dimpled. Two years before admission he noted progressive weakness of the right half of the face, blurred vision on looking toward the left, decreased hearing on the right, and occasional intense pain in the base of the skull on rising from a stooping position or on sudden turning.

A year before admission he began to have daily parietal headaches on either the right or the left side. Sometimes these would last all day and were of sufficient severity to make him turn pale, break out in a cold sweat, and grasp his head in both hands. At such times he might not be able to see well on looking toward the left. For the month before admission, however, the headaches had been less troublesome.

Ear, nose, and throat examination revealed the velum of the palate to be higher on the right than on the left, but the results were negative otherwise except for findings in regard to function of the right inner ear. Hearing for pure tones at threshold was normal in the left ear. On the right, however, testing at various frequencies gave the following findings in decibels:

	128	256	512	1024	2048	4096
A. C.	25	25	30	35	20	20
B. C.	..	25	35	35	20	25

The loudness balance test revealed slight and incomplete recruitment at frequencies of 512, 1024, and 2048 cps. The narrowed dynamic range for hearing on the right was demonstrated by the 25 db. difference between the speech reception threshold and maximal comfort level on the right as compared with 45 db. on the left.

The discrimination test for phonetically balanced words using the Central Institute for the Deaf recordings showed a 95% discrimination on the right at 50 db., whereas on the left at 15 db. above threshold a maximal discrimination of 65% was obtained, which dropped to 55% at a further increase of 10 db.

At operation an astrocytoma, Grade 2, was found which had invaded the right internal auditory meatus from the cerebellopontine angle.

When greater pressure had been caused by firmer and larger growths, Eby and Williams found even partial recruitment to be absent.

Comment.—There are many difficulties and pitfalls in the diagnosis of Ménière's disease without hydrops, Ménière's disease with endolymphatic hydrops, and cochlear deafness or endolymphatic hydrops without vertigo. Cases 1 to 7 indicate some of the difficulties that may be experienced. There is some indication that these exceptional cases may have similar end-phenomena to those of Ménière's disease but resulting from a somewhat different basic physiologic mechanism. Perhaps some term ought to be found for this group other than endolymphatic hydrops, such as the "hydrolabyrinth" suggested by Brunner.

In the usual cases of endolymphatic hydrops differentiation is rather readily made, as Dix, Hallpike, and Hood [41] showed by the loudness balance test and by the discrimination test for phonetically balanced words. This finding has recently

41. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Observations upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve, J. Laryng. & Otol. **62**:671-686 (Nov.) 1948.

been confirmed by Eby and Williams.[42] In such a case symptoms are unilateral to a considerable extent at least, although Cawthorne, Fitzgerald, and Hallpike [43] were able to demonstrate slight cochlear changes in the better, or "unaffected," ear in more than 80% of cases of Ménière's disease. It is my opinion, therefore, that the loudness balance test and the percentage reception curve for phonetically balanced words are the best guides and that when hydrops of any degree is actually present hypoactivity in the involved equilibrial labyrinth will be found.

TREATMENT

If a definite diagnosis of Ménière's disease can be made, not only relief from the vertiginous crises but also restoration of hearing can usually be secured if the patient can and will adhere to the regimen described. Many of these patients are subject to anxiety and tension states. Relief of these conditions is of great importance in a favorable clinical result.

Since Ménière's disease appears to be an expression of an inherited constitutional tendency, it would be expecting too much of any therapeutic plan to eradicate such a tendency. Therefore, even after symptoms have been relieved recurrences are to be expected. Few patients are so situated that they can devote their entire time to control of this disorder. Treatment, therefore, should be designed with the well-being and maximal convenience of the patient uppermost in mind.

Successful medical treatment of Ménière's disease has been based on the hypothesis that the condition is due to a localized dysfunction of the normal autonomic defense mechanism which includes the peripheral vascular bed, in other words, an allergy. So-called specific therapy, such as elimination of foods and contacts or specific desensitization, however, has successfully relieved the disorder in a few cases. Such immunologic reactions may be a continually recurring precipitating factor, and the possibility of their presence should not be ignored. In my experience, however, the more frequent precipitating factor is a physical stimulus.

Therefore, use of a logical combination of therapeutic agents known to affect the various factors operating in physical allergy seems reasonable, rather than use of one and then another agent in tandem. As Williams, Horton, and Day [40] reported in a previous paper, the first measurable evidence of the restoration of hearing may not appear until after two to four weeks of vigorous treatment. Therefore therapeutic trial should not be given up too early.

Vasodilators.—The use of vasodilators to relieve the arteriolar spasm present in allergy is one of the first methods of treatment suggested.

In my hands monoethanolamine nicotinate (nicamin®) has been found the simplest, easiest, and least time-consuming vasodilator to use from the point of view of both patient and physician. This drug is given subcutaneously, the initial dose being 25 mg. If this dose is well tolerated, the quantity is increased by an equal amount at the daily or twice daily injection until the optimal dose, usually 100 mg. a day, is reached. The patient is instructed in the technic of self-medication and

42. Eby, L. G., and Williams, H. L.: Recruitment of Loudness in the Differential Diagnosis of End Organ and Nerve Fibre Deafness, Tr. Am. Laryng. Rhin. & Otol. Soc. **55**:134-148, 1951; Laryngoscope **61**:400-414 (May) 1951.

43. Cawthorne, T. E.; Fitzgerald, G., and Hallpike, C. S.: Studies in Human Vestibular Function: III. Observations on the Clinical Features of "Ménière's" Disease, with Especial Reference to the Results of the Caloric Tests, Brain **65**:161-180 (June) 1942.

told to continue treatment at home. The injections are continued until the maximal possible improvement in hearing is obtained. The dose is then decreased by increasing the interval between injections. Injections are given every other day for two weeks, three times a week for one week, twice a week for one week, and then stopped. If at any subsequent time symptoms begin to return, treatment is again started at 25 mg. and built up to 100 mg. and continued there for one week. Such booster courses are required by the average patient two or three times a year.

For some patients it is possible to substitute nicotinic acid tablets for the monoethanolamine nicotinate. Fifty to 100 mg. once or more daily can be given. Clinical improvement in some patients, however, does not continue when oral administration is used. Beta-pyridylcarbinol tartrate (roniacol®) given by mouth also has proved to be an effective vasodilator in many instances.

Diet.—Kern [17] stated that changes in the water balance of the body may influence the occurrence of allergic phenomena. Increased intake of sodium by tending to retain interstitial fluid, a sort of sodium brine being formed, also will favor the development of allergic reactions. Increased intake of potassium or decreased intake of sodium by tending to increase intracellular fluid and decrease interstitial fluid, will, on the other hand, antagonize allergic reactions. Beckman [44] considered allergy as a special type of alkalosis. He defined potential alkalosis as the tendency of an individual to store up too much alkali reserve without necessarily having clinical alkalosis. He considered that potential alkalosis is an important causative factor in allergy. As one method of treatment he suggested that patients with an allergy eat fully of fats, meats, and cereals, while green vegetables and fruits should be avoided.

Schemm [45] proposed an ambulatory diet which was not merely a low-salt or an acid- or neutral-ash diet, but a combination of all. This diet, modified somewhat, is as follows:

Maximal Intake in 24 Hr.		Minimal Intake in 24 Hr.	
Food	Amount	Food	Amount
Milk	1 pint	Eggs	2
Vegetables	2 servings	Meat, fowl, fish	2 servings
		Cereal foods	Equivalent to 6 slices of bread
Fruit	2 servings except for ⟶	Prunes, Plums, Cranberries	As desired

1. Avoid adding table salt to food and avoid foods prepared with salt, such as pickled herring or sauerkraut.
2. Avoid soda, such as baking soda and soda biscuits, and be careful of artificially softened water and "alkali" water.
3. Avoid salty broths and soups.

I have found that patients can be persuaded with relative ease to continue to use this diet. Restriction of total intake of fluid to about 3,000 cc. daily has been found advisable, together with the use of a diuretic to increase elimination of fluid and sodium. Ammonium chloride in divided doses of 6 to 9 gm. daily has the additional advantage of producing a relative acidity of the tissue fluids.

Methantheline Bromide (Banthine®).—Since allergy appears to be associated with cholinergic preponderance, the use of a drug inhibiting the cholinergic fibers

44. Beckman, H.: Allergy Considered as a Special Type of Alkalosis, J. Allergy **1**:496-500 (Sept.) 1930.

45. Schemm, F. R.: A High Fluid Intake in the Management of Edema, Especially Cardiac Edema: I. The Details and Basis of the Régime, Ann. Int. Med. **17**:952-969 (Dec.) 1942.

of the autonomic nervous system seems indicated in Ménière's disease. Methantheline bromide (banthine®) acts as a blocking agent in the ganglia of the autonomic system. It also has an atropine-like action on the nerve endings of the cholinergic fibers. For this reason, its action in therapeutic doses is largely on the cholinergic nerves.

As a full therapeutic dose, 100 mg. of methantheline bromide every six hours, or 50 mg. every four hours (night and day), is recommended. After symptomatic relief has been obtained the dose may be reduced to 50 mg. every six hours as a maintenance dose. Since stress appears to be an important factor as a precipitating stimulus in Ménière's disease, it is important that the patient be advised to increase his dosage from the maintenance level to the therapeutic level for the duration of any emotional or other stress.

In my hands, methantheline bromide has been most effective in improving hearing in cases of Ménière's disease with endolymphatic hydrops.

Duration.—It is felt that the best results are obtained in endolymphatic hydrops by a combination of these three types of therapy. This combination consists in the elimination of sodium with an attempt to produce a relative acidosis, the use of a vasodilator to "unlock" the arteriolar spasm and sweep toxic metabolites into the general circulation, and methantheline bromide to depress the overactive cholinergic nerves. It is important not to expect too rapid improvement in the cochlear symptoms produced by the hydrops. In a few instances definite improvement has not occurred for four to six weeks after this regimen was started. That improvement may and does eventually occur with treatment is evidenced in the following case.

CASE 8.—A man aged 42 years stated that a year before admission to the clinic buzzing began in his right ear. Three months later a severe attack of rotatory vertigo, nausea, and vomiting lasting two hours occurred. Six months before admission he had a second similar attack which was followed by several attacks each week except for a week of freedom two months before admission. He had been dizzy for as long as three weeks at a time.

The hearing in the right ear had been variable during this entire period but on the whole had grown gradually worse. He had found that violent purgation would give considerable subjective relief of symptoms, particularly the deafness. He had many inflations of his Eustachian tubes, without relief. He had taken dimenhydrinate, 50 mg. three times a day, for several months. This had prevented vomiting but had not affected his disorder otherwise.

Ear, nose, and throat examination gave negative results except for the findings in regard to the hearing and labyrinthine function. Hearing in the left ear for pure tones at threshold was essentially normal. Hearing in decibels in the right ear for pure tones of various frequencies at threshold showed perception deafness (Charts 3 and 4).

Loudness balance tests showed hyperrecruitment in the right ear over the left at frequencies of 512, 1024, and 2048 cps. Testing the function of the equilibrial labyrinth by a modification of the Kobrak test showed marked hypoactivity in the right labyrinth.

This patient received a modification of the Schemm low-sodium acid to neutral-ash diet, 9 gm. of ammonium chloride daily in divided doses, 50 mg. of methantheline bromide every four hours night and day, and 100 mg. of monoethanolamine nicotinate by subcutaneous injection daily. On this regimen he showed no objective improvement in the hearing for 11 days and only a slight improvement in his subjective symptoms. The audiometric findings on the 10th day are shown in Charts 4 and 5.

Loudness balance tests still showed hyperrecruitment in the right ear over the left. Improvement, although slight, was definite, however. The patient was advised to continue treatment at home and to return for observation in two weeks.

On his return he stated that he had been able to work during the previous two weeks for the first time in seven months and that his attacks were infrequent and more like sick headaches than crises of Ménière's disease (Charts 6 and 7).

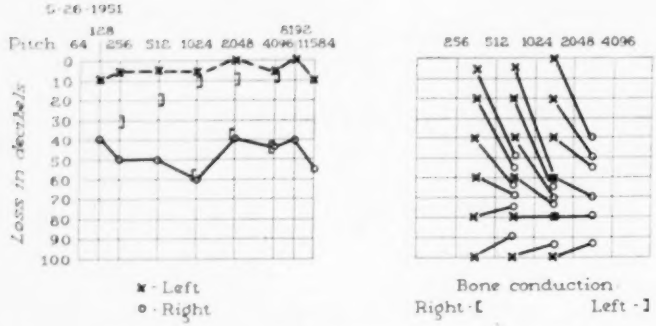

Chart 3 (Case 8).—The audiometric findings on the patient's admission indicated an endolymphatic hydrops in the right ear although the discrimination deafness (Chart 4) was not as great as is often found.

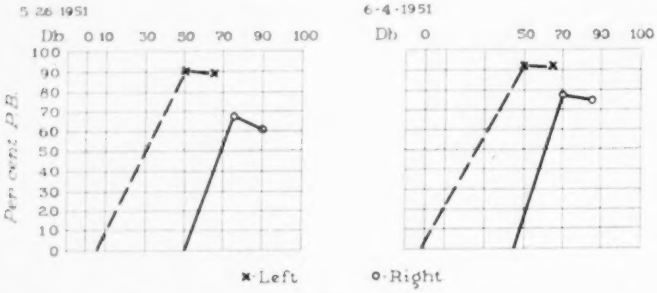

Chart 4 (Case 8).—The discrimination deafness was not as great in the right ear on the patient's admission (May 26, 1951) as it often is in endolymphatic hydrops. By June 4, 1951, there was slight improvement of the discrimination deafness.

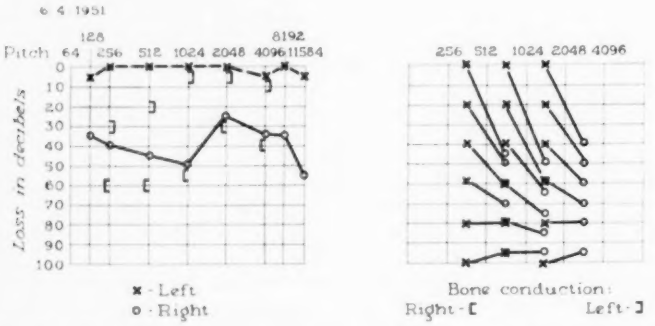

Chart 5 (Case 8).—Little improvement is shown after ten days of treatment although a slight improvement of the discrimination deafness may be noted (Chart 4).

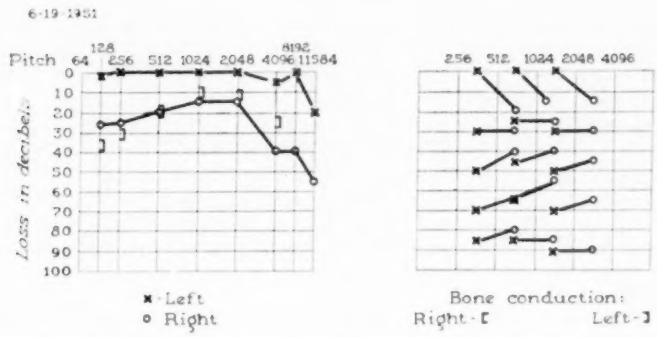

Chart 6 (Case 8).—Definite audiometric evidence of improvement is shown after about three weeks of treatment.

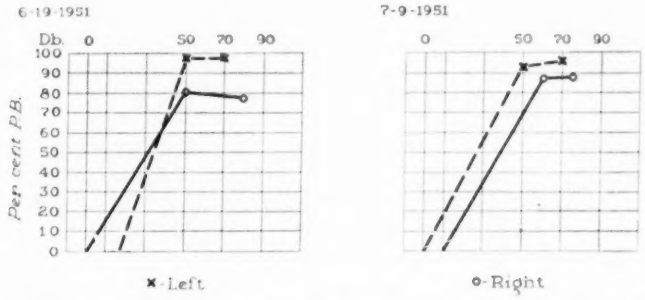

Chart 7 (Case 8).—Discrimination deafness had improved after three weeks of treatment and more after five weeks of treatment.

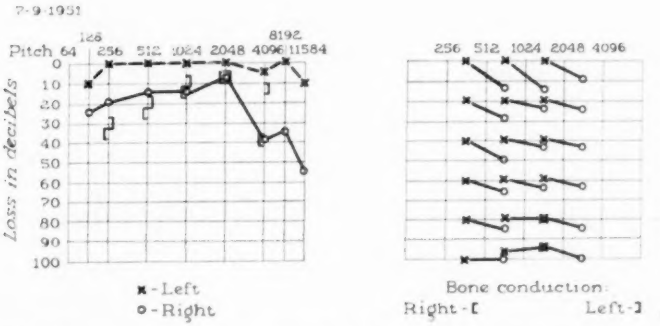

Chart 8 (Case 8).—The audiometric findings after about five weeks of treatment show slow but steady improvement.

Loudness balance tests showed complete recruitment of the right ear at only 30 db. below threshold of the left ear and hyperrecruitment at 50 db.

Speech reception tests done with recordings from the Central Institute for the Deaf showed an improvement in recognition of phonetically balanced words from 65 to 75%. The patient was again sent home to continue treatment for another two weeks. On his return audiometric findings showed improvement (Charts 7 and 8).

Speech reception tests showed a discrimination in the right ear of 75% for phonetically balanced words. The patient was again sent home with instructions to continue the treatment. Chart 9 shows the audiometric findings one and two months later.

The patient had been completely free of any symptoms of vertigo, unsteadiness, dizziness, tinnitus, or deafness for the past two months. He was advised to continue with his diet but to decrease the dose of methantheline bromide to 50 mg. every six hours and to discontinue the use of ammonium chloride and monoethanolamine nicotinate. He was asked to return for a recheck of his hearing in a month.

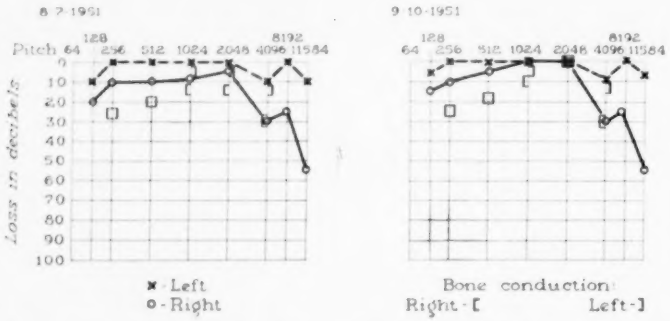

Chart 9 (Case 8).—The threshold audiograms for pure tones by air and by bone conduction are shown after three and after four months of continuous treatment.

This case illustrates the rather slow progress that can be obtained even with vigorous therapy but shows that if treatment is persisted in successful results can often be obtained.

SUMMARY AND CONCLUSIONS

The physiologic basis of allergy of the inner ear, usually termed Ménière's disease, is considered in its several variations. These are autonomic dysfunction with its stereotyped or invariable reaction on the peripheral vascular bed, resulting in transudation of an excess fluid high in protein content and consequent elevated osmotic pressure. The vestibular symptoms seem to be mainly due to vasospasm, while the cochlear symptoms appear to depend in the main on an increase in endolymph of high osmotic pressure.

The importance of, and some of the pitfalls in, differential diagnosis are stressed, and some illustrative case reports are reviewed. The treatment which has given the most satisfactory results at the Mayo Clinic has been a modification of Schemm's diet with other measures to help in the elimination of sodium, administration of a vasodilator to decrease arteriolar spasm, and administration of an inhibitor to depress overactive cholinergic nerves.

NEOPLASM OF THE NASOPHARYNX

Review of Sixty-Eight Cases Which Appeared in the Japanese Literature

TOSHIZO DAITO, M.D.
HIDEO SAKAMOTO, M.D.
TOKYO, JAPAN
AND
H. JAMES HARA, M.D.
LOS ANGELES

YAMASHITA, in 1941, recorded 29 cases of malignant neoplasm that were observed in one of the university hospitals in Formosa. Of these, 4 were in Japanese and 25 were in Chinese. The total number of patients treated in this university hospital for all causes was 12,722 Japanese and 7,756 Formosans, the proportion being nearly 3:2. Since the Japanese and Chinese are supposed to have a common racial origin, this marked susceptibility of the Chinese is an interesting phenomenon.

This review covers the 68 cases of nasopharyngeal tumors that appeared in the Japanese medical literature since the above-mentioned publication. There are two major types of nasopharyngeal growths. One originates in the nasopharynx; the other is an extension from the base of the skull, and the tumor appears secondarily in the nasopharynx. The presenting symptoms of these two types are much the same. This paper is chiefly concerned with the clinical manifestations.

STATISTICS

In tabulation of the reported cases, lymphoepithelioma of Schmincke is placed under carcinoma, and small-cell sarcoma is classified under sarcoma. Since endotheliomas are malignant growths of nonepithelial origin, they are grouped under sarcoma. We are not unaware that in the light of the prevailing concept of malignant neoplastic diseases in this area such a classification may be disputed by some. Table 1 indicates the distribution of tumors according to cytology as originally reported. There were 42 males (62%) and 24 females (35%). For two patients the sex was not stated. There were 1.7 times as many males as females.

Carcinoma.—Table 2 indicates the types of carcinoma in 33 cases of neoplasm of the nasopharynx.

Table 3 indicates the age distribution in 33 cases of nasopharyngeal carcinoma.

Sarcoma.—Table 4 indicates the various forms of nasopharyngeal sarcoma recorded.

Table 5 indicates the age distribution in 32 cases of nasopharyngeal sarcoma.

Dr. Daito is Professor and Dr. Sakamoto, Assistant, Department of Otolaryngology, Nippon Medical School. Dr. Hara is from the Department of Otolaryngology, College of Medical Evangelists.

TABLE 1.—*Cytologic Distribution of Neoplasms of Nasopharynx*

	No. of Cases	% of Cases
Carcinoma	33	48.5
Sarcoma	32	47.1
Myxosarcoma endothelioma	1	1.5
Not clearly stated	2	2.9
	68	100.0

TABLE 2.—*Types of Carcinoma*

Classification	Total	%	Male		Female	
			No.	%	No.	%
Epithelioma	10	30.3	5	15.2	5	15.2
Carcinoma (unclassified) *	10	30.3	7	21.2	2	6.1
Basal-cell carcinoma	6	18.2	3	9.1	3	9.1
Lymphoepithelioma	6	18.2	5	15.2	1	3.0
Malignant papilloma	1	3.0	1	3.0	0	0

* Sex of one patient was not stated.

TABLE 3.—*Age Distribution in Cases of Carcinoma*

Age, Yr.	Total No.	%	Male		Female	
			No.	%	No.	%
11-20	3	9.1	2	6.1	1	3.0
21-30	3	9.1	1	3.0	2	6.1
31-40	6	18.2	5	15.2	1	3.0
41-50	7	21.2	3	9.1	4	12.1
51-60	11	33.3	7	21.2	4	12.1
61-70	2	6.1	1	3.0	1	3.0
Unknown	1	3.0	1	3.0	..	...

TABLE 4.—*Types of Nasopharyngeal Sarcoma*

Types of Sarcoma	Total No.	%	Male		Female	
			No.	%	No.	%
Round-cell sarcoma	9	28.1	3	9.4	6	18.8
Reticulum-cell sarcoma	7	21.9	6	18.8	1	3.1
Lymphosarcoma	4	12.5	2	6.3	2	6.3
Polymorphous sarcoma *	2	6.3	1	3.1	0	0
Spindle-cell sarcoma	2	6.3	1	3.1	1	3.1
Sarcoma (recorded as such)	4	12.5	4	12.5	0	0
Hemangioendothelioma	1	3.1	1	3.1	0	0
Lymphoendothelioma	1	3.1	0	0	1	3.1
Meningioendothelioma	1	3.1	0	0	1	3.1
Endothelioma (unclassified)	1	3.1	0	0	1	3.1

* Sex of one patient was not stated.

TABLE 5.—*Age Distribution in Cases of Sarcoma*

Age, Yr.	Total No.	%	Male		Female	
			No.	%	No.	%
1-10	1	3.1	1	3.1	0	0
11-20	5	15.6	4	12.5	1	3.1
21-30	7	21.9	2	6.3	5	15.6
31-40	3	9.3	2	6.3	1	3.1
41-50	7	21.9	3	9.3	4	12.5
51-60	5	15.6	3	9.3	2	6.3
61-70	2	6.3	2	6.3	0	0
71-80 *	2	6.3	1	3.1	0	0

* Sex of one patient was not stated.

CLINICAL OBSERVATIONS

The primary site of malignant growth is not always easy to determine. This is particularly true when the growth occupies more than one-half of the nasopharynx. Table 6 indicates the primary site of neoplasm in the 32 cases in which it was clearly stated. Thus, there were 19 cases of invasion of the roof, base, and either one or the other midportion of the nasopharynx. These appeared to be the sites of predilection. There were 11 cases in which growths were limited to the lateral walls of the nasopharynx, the second most frequent site.

SYMPTOMATOLOGY

In a majority of instances the growth was well established in the nasopharynx by the time patients consulted the physician. When the growth involved various cranial nerves, the prognosis was extremely grave.

TABLE 6.—*Primary Site of Nasopharyngeal Neoplasm in Thirty-Two Cases*

Primary Site	No.	%
Right Eustachian tubal orifice and fossa of Rosenmüller	5	15.6
Left Eustachian tubal orifice and fossa of Rosenmüller	6	18.8
Right half of nasopharynx and its roof	3	9.4
Left half of nasopharynx and its roof	9	28.1
Base of nasopharynx	7	21.9
Posterior wall of nasopharynx	2	6.2

TABLE 7.—*Nerves Involved in Nasopharyngeal Neoplasm in Order of Frequency*

1. Abducens	7. Oculomotor
2. Trigeminus	8. Spinal accessory
3. Hypoglossal	9. Trochlear
4. Vagus	10. Acoustic
5. Glossopharyngeal	11. Optic
6. Facial	

In a number of the cases reported from the Yamashita clinic in Formosa there were complaints of nasal obstruction, impaired hearing, tinnitus, involvement of the trigeminal nerves, and anemia. A number of reports in this review indicated the presence of cervical adenopathy. Among 68 cases, there were 36 (52.9%) with intracranial involvement. The following nerves were involved in order of frequency:

In most cases, multiple nerves were affected rather than a single nerve. With regard to the type of lesions, carcinoma was found in 19 cases and sarcoma in 15. The type of lesion was unknown in two cases.

The cranial nerves were affected in 27.9% of the cases of carcinoma and in 22.1% of those of sarcoma, constituting, respectively, 57.5 and 46.8% of the total cases reported. The pseudo-Gradenigo, Horner, Jacob, and Avellis syndromes and Trotter triads were recorded in some instances.

TREATMENT

Whenever accessible, surgical extirpation of the growth after Hoshino's technique was followed. This consists of splitting the soft palate, which permits an

easier inspection of the growth and a direct approach to the site of the neoplasm. This was followed by radium implantation and deep x-ray therapy. In inoperable cases only radium and x-ray treatments were given.

PROGNOSIS

Prognosis is poor for malignant neoplasm of the nasopharynx. In this series of 68 cases, cure was reported in 10 and improvement in 7. There were 28 deaths and 23 cases of unknown outcome. The longest survival from the onset of the disease was five years; the shortest was 6 months, the average being 16 months.

CHOLESTEATOMA IN RADICAL MASTOIDECTOMY CAVITY SECONDARY TO USE OF PRIMARY SKIN GRAFTS

N. VERN PETERSON, M.D.
RYE, N. Y.

CHOLESTEATOMA of the middle ear and mastoid process has been the subject of much study and speculation since it was first described by Müller,[1] in 1838, and since Virchow [2] gave the first explanation of its origin, in 1855. It is the purpose of this paper to present evidence of cholesteatoma formation following the use of skin grafts in the cavity resulting from radical mastoidectomy, to correlate these findings with the theories of the histogenesis of cholesteatoma, and to question strongly the procedure of skin grafting after a radical mastoidectomy.

Prior to 1946 I had performed 268 radical mastoidectomies. The technique used in all these cases was the classic postauricular radical mastoidectomy procedure. The operation was carried out by means of the gouge, chisel, curette, and rongeur. The mastoid cavity and the epitympanic and hypotympanic spaces were rather carefully cleaned out. The tympanic space was cleansed of the grosser pathological tissues, but the area of the round and oval windows was not greatly disturbed. Any diseased bone overlying dura in any location was carefully removed, leaving a healthy bony margin. A Ballance type of flap was used, and the cavity was well packed with iodoform or petrolatum gauze. The postoperative care consisted of frequent removals of packing, cleansing of the wound, and repacking. Any excessive granulations were removed or cauterized, as seemed indicated. In later years Sulzberger's weak iodine powder and then sulfanilamide or sulfathiazole powders were used without packing and repacking. Under this operative and postoperative regimen it has been my experience to encounter cases several years after operation with superficial infections and granulations, but I have never seen any evidence of cholesteatoma in these old mastoid cavities. It is quite possible that in some of these cases there were small accumulations of cholesteatoma, but the amount was so insignificant as to escape my observation.

This technique of radical mastoid surgery was abandoned in 1946 in favor of the endaural approach. After a special course in endaural surgery with Dr. Julius Lempert, I became convinced that this technique offered a real advance in mastoid surgery. It is my feeling that this training not only opened up a new and better approach to the disease process in chronic mastoiditis but also made me a more

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1. Müller, cited by Lederer, L. L.: Diseases of the Ear, Nose, and Throat; Principles and Practice of Otorhinolaryngology, Ed. 3, Philadelphia, L. A. Davis Co., 1942.

2. Virchow, cited by Politzer, A.: Diseases of the Ear; revised by M. J. Ballin, Ed. 6. Philadelphia, Lea and Febiger Co., 1926.

careful, more observant, and more painstaking operator. Accordingly, I approached this problem of extensive mastoid surgery with a new confidence and great expectations. For the most part, I have followed carefully the technique as outlined by Lempert,[3] occasionally modifying my technique according to the method of House.[4] Since 1946 I have performed 52 endaural radical mastoidectomies.

The basic change in my operative procedure consisted in a new approach and, particularly, a much more complete removal of the diseased tissue. With the employment of the electrically-driven burr and magnification of the binocular loupe, the tympanic cavity was now scrupulously cleansed of all diseased tissue. Following the technique of House,[4] the periosteum of the antrum, attic, and tympanic cavity with its overlying granulations and debris was carefully elevated under magnification and removed completely. The areas of the round and oval windows which had previously been avoided were now thoroughly débrided. In all cases an attempt was made to remove the cholesteatoma matrix as completely as possible. Primary skin grafting was employed in 41 of the 52 cases. At first these grafts consisted of small pieces of the external membranous canal wall. Later full-thickness grafts were taken from the thin postauricular skin, or split-thickness grafts were taken from the inner aspect of the thigh, and the tympanic cavity, attic, and mastoid process were almost completely covered. These grafts were held in place by pledgets of parresined-gauze mesh for a period of six to eight days. No further packing was used in the postoperative care, and an attempt was made to avoid touching the grafts or the inside of the cavity except by the insufflation of Sulzberger's or sulfonamide powders.

In general, I feel that the results obtained by the endaural method have been far superior to those obtained by the old postauricular technique. Patients have had much less discomfort, the period of hospitalization has been shorter, and the cosmetic result has been much better. In the majority of cases the cavities have healed more rapidly; there have been fewer granulations, and the result has been a dry, smooth cavity lined with healthy epithelium. However, there have been a number of cases in which healing was apparently progressing satisfactorily when suddenly the whole cavity filled with granulation tissue, requiring months of care. This is an occurence not infrequently observed by all those doing mastoid surgery. As noted also by other men,[5] this has happened in cases in which I felt that I had done a very thorough mastoidectomy and justifiably expected rapid healing and a dry cavity. In my experience the majority of these patients have done better under conservative treatment. The granulations often start receding as suddenly as they commenced, more or less uninfluenced by local treatment. Two such patients in whom extensive granulation tissue developed were unable to return for routine treatments. They received no treatment whatsoever and were found six to nine months later to have clean, solid, dry cavities.

3. Lempert, J.: Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. **27**:555-587 (May) 1938. Lempert, J.: Endaural Subcortical Mastoidotympanectomy for the Cure of Chronic Persistent Suppurative Otitis Media, Arch. Otolaryng. **49**:20-35 (Jan.) 1949.

4. House, H. P.: Surgery of the Chronically Discharging Ear, Arch. Otolaryng. **49**:135-150 (Feb.) 1949.

5. McQuiston, R. J.: Endaural Radical Mastoidectomy for Chronic Mastoiditis, Arch. Otolaryng. **51**:596-607 (April) 1950.

Of the 41 cases in which skin grafts were used, in 11 cases varying amounts of cholesteatomatous material developed in the cavities from one to four years after operation. "Cholesteatomatous material" is here used to designate the typical clinical entity recognized by every practicing otologist. There was nothing unusual about these 11 cases at operation. Just as in the other 41 cases, a very complete mastoidectomy was carried out, with removal of all cholesteatomatous matrix. In each case healing appeared to be rapid and quite satisfactory for the first six to eight weeks. Then the cavities appeared to fill with granulation tissue which was covered by a smooth "false" membrane. In some instances this process extended out to the external meatus. Eventually in each case this membrane either broke down spontaneously or was incised to release a contained mass of cholesteatomatous material. In all these cases the quantity was not insignificant, and the material obtained could not be distinguished grossly from that regularly seen in chronic mastoiditis with cholesteatoma formation. In two of my patients these masses of cholesteatoma were positively alarming, and it was felt that there was definite bony erosion occurring in the operative field. As yet, in none of these cases has a secondary operation been required, but at the present time I have under treatment one of my skin-graft patients who was operated upon two years ago. So far I have been unable to remove all the cholesteatomatous material which has formed, and I feel that it will be necessary to do a secondary operative procedure.

REPORT OF CASES

Following are abstracts of four typical cases.

CASE 1.—J. I., a 54-year-old white woman, first seen in February, 1947, had a history of a discharging right ear since childhood. In the past month pain had commenced on the right side of her head and she had had persistent vertigo. Examination revealed a large attic perforation of the right ear with granulation, cholesteatoma, and heavy purulent secretion. Findings on the general physical examination were essentially negative. On April 10, 1947, a radical endaural mastoidectomy was performed. Granulations and cholesteatoma were found throughout the middle ear cavity, epitympanum, and mastoid antrum, with erosion through the plates of the tegmen antri and lateral sinus. All diseased tissues and cholesteatoma with its matrix were removed. Skin grafts taken from the membranous canal wall were used to line the finished cavity. The postoperative course was uneventful, and the wound appeared healed on June 17. She was seen at infrequent intervals until July 23, 1948, at which time the cavity was dry and clean. On May 17, 1951, she was seen with a history of pain and discharge for the past few months from the ear which had been operated upon. She was also having a considerable amount of vertigo. On examination the mastoid cavity was found to be almost completely closed by a fibrous membrane, behind which there was a large mass of cholesteatoma, granulations, and purulent secretion. When this was evacuated, it was my impression that definite bony erosion had taken place. The cavity was thoroughly cleaned, cauterized with silver nitrate, and packed with petrolatum gauze. After approximately one month of treatment, the cavity was again completely healed and covered with healthy epithelium.

CASE 2.—M. F., a 17-year-old white youth, first seen in October, 1947, had a history of a discharging right ear for the past 10 years. Findings on a general physical examination were within normal limits. The nose and throat were essentially normal except for a moderate amount of lymphoid tissue in the nasopharynx. There was a large posterior marginal perforation of the right ear, with granulations and cholesteatoma. The fistula test was positive. On March 23 a right endaural radical mastoidectomy was performed. A large amount of bony destruction was encountered, with granulations and cholesteatoma filling the enlarged mastoid antrum, aditus, and attic. A careful exenteration was carried out, with removal of the cholesteatomatous matrix. A full-thickness skin graft taken from the postauricular area was used

to line the cavity. Healing was uneventful, and soon afterward this patient entered the Army. He was next seen in September, 1949, at which time the middle ear was found to be closed off by a fibrous membrane. This membrane was excised and a large mass of cholesteatomatous material was revealed. Treatment was required for the next five months. When the patient was seen recently there was no evidence of recurrence.

CASE 3.—J. D., a 42-year-old white woman, was first seen Aug. 17, 1948. She gave a history of frequent headaches and a discharging left ear for the past two months. Examination at this time revealed an attic perforation of the left ear, from which protruded an aural polyp; behind this was a large mass of cholesteatomatous material. General physical examination was noncontributory. On Aug. 22, 1948, a left endaural radical mastoidectomy was performed. The middle ear cavity was full of granulations and cholesteatoma extending into the attic and mastoid antrum and eroding through the dural plate in the region of the tegmen tympani. A careful exenteration of all diseased tissue was carried out, and under magnification granulations and cholesteatoma were removed from the middle ear cavity and mastoid antrum. A skin graft taken from the external membranous canal wall was applied to the tympanic space and mastoid antrum area. Healing was uneventful and rapid. She was seen at infrequent intervals for the next two years. On Aug. 20, 1951, she came to the office, complaining of discharge and pain during the past few weeks in the ear which had been operated upon. At this time a large portion of the cavity was found to be occluded by a fibrous membrane. There was a small opening in this membrane, leading into the tympanic space, from which exuded foul-smelling purulent secretion. When this opening was enlarged by incision, an extensive mass of cholesteatomatous material was removed. Granulations were cauterized with silver nitrate, and the cavity was packed with petrolatum gauze. This patient is progressing satisfactorily but is still under treatment.

CASE 4.—R. O., a 35-year-old white man, was first seen as a child in 1928 with an acute mastoiditis in the right ear. He recovered from this after a myringotomy, and no mastoid surgery was required. He was seen for the next five years, during which time he had occasional bouts of acute otitis media, particularly in the right ear. There was a persistent posterior-superior perforation of the right drum head. In 1935 his right ear showed almost complete destruction of the tympanic membrane and a cholesteatoma of the tympanum and antrum. Operation was refused at this time, and he was treated conservatively until Feb. 17, 1947, when a radical endaural mastoidectomy was done. It was found that extensive bony destruction had occurred, forming a large cavity out of the mastoid antrum and surrounding cells. The tympanic cavity and attic were filled with granulations and cholesteatoma. After a complete exenteration of the diseased bone and removal of all cholesteatoma and granulations, the cavity was lined with skin taken from the external membranous canal. Immediate healing was good, but the cavity soon became almost completely filled with granulations, which persisted in spite of all forms of local treatment for the next two years. On March 23, 1950, this patient presented himself with an acute exacerbation of the ear which had been operated upon. Under local anesthesia the bulk of the superficial granulation tissue was removed, and a mass of cholesteatomatous material was removed. This was approximately ½ in. (1 cm.) in diameter. From this time on the remaining granulations rapidly disappeared, the cavity became dry and within a month was lined with healthy epithelium. At present, the cavity is dry and appears to be completely healed.

My experience in these 11 cases has led me to search the literature for reports of similar findings by other otologists and to review the theories of the histogenesis of cholesteatoma. Much has been written concerning the conflicting views on the theory of the etiology of cholesteatoma, but nowhere could I find evidence of cholesteatoma formation from buried epidermis.

The term "cholesteatoma" is actually a misnomer, as has been brought out by Eggston and Wolff.[6] "Oma" signifies a neoplasm or autonomous new growth of

6. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose, and Throat, Baltimore, Williams & Wilkins Company, 1947, pp. 202, 259, 336, 424-434.

tissue. Bezold and Siebenmann [7] were the first to consider that cholesteatoma was not a true neoplasm but was the result of an inflammatory process. Even the prefix is misleading, as was pointed out by Dean and Pfingsten.[8] They showed that normal cerumen has been found to contain more cholesterol, both in the amorphous and crystalline forms, than does a cholesteatoma. According to Lederer,[9] the term cholesteatoma was first used by Müller in 1838. It has been defined as a tumor consisting of a thin shell of epidermis and connective tissue inside of which is accumulated desquamated horny epithelium. Morrison [10] defines it as "a mass of layers of cast-off epithelial cells with fatty degeneration and cholesterin crystals on a matrix of subepithelial connective tissue with a squamous-epithelial covering." Tumarkin [11] denied the presence of prickle cells in the lining of a cholesteatoma. This has been refuted by Eggston and Wolff,[6] and, recently, Begley, McDonald, and Williams [12] presented microscopic evidence of prickle cells in the epithelial layer of an aural cholesteatoma and established that it was true epidermis.

In a recent paper Begley, McDonald, and Williams [12] have made a very complete review of the literature covering the subject of histogenesis of cholesteatoma, and a certain amount of repetition is unavoidable. There are three strongly supported theories of the origin of cholesteatoma, namely: (1) the primary, or neoplasm, theory; (2) The invasion, or immigration, theory, and (3) the metaplasia theory.

1. The primary, or neoplasm, theory is based on the belief that cholesteatoma is a true tumor arising from congenitally misplaced anlage. Eggston and Wolff [6] felt that this was a relatively rare occurrence and that the terms primary and true cholesteatoma should be used only for those tumors arising from embryonic cell rests. Holmes,[13] Diamant,[14] McKenzie,[15] Grove,[16] Teed,[17] Lindsay,[18] and Freedman

7. Bezold, F., and Siebenmann, F.: Textbook of Otology for Physicians and Students, translated by J. Holinger, Chicago, E. H. Colegrove Co., 1908.

8. Dean, L. W., Jr., and Pfingsten, M. G.: A Chemical and Cytologic Study of Aural and Nasal Exudates as Regards the Chemical Diagnosis of Cholesteatoma, Ann. Otol. Rhin. & Laryng. **43**:484-497 (June) 1933.

9. Lederer, F. L.: Diseases of the Ear, Nose and Throat: Principles and Practice of Otorhinolaryngology, Ed. 3, Philadelphia, F. A. Davis Company, 1942.

10. Morrison, W. W.: Diseases of the Nose, Throat, and Ear, Philadelphia, W. B. Saunders Company, 1938, pp. 488, 568-570.

11. Tumarkin, I. A.: A Contribution to the Study of Middle Ear Suppuration with Special Reference to the Pathogeny and Treatment of Cholesteatoma, J. Laryng. & Otol. **53**:685-709 (Nov.), 737-771 (Dec.) 1938.

12. Begley, J. W., Jr.; McDonald, J. R., and Williams, H. L.: Histology and Histogenesis of Cholesteatoma of the Middle Ear and Mastoid, A. M. A. Arch. Otolaryng. **53**:41-52 (Jan.) 1951.

13. Holmes, E. M.: A Review of 303 Cases of Cholesteatoma, Ann. Otol. Rhin. & Laryng. **47**:135-143 (March) 1938.

14. Diamant, M.: Cholesteatoma and Chronic Otitis: Etiologic Problems, Arch. Otolaryng. **47**:581-589 (May) 1948.

15. McKenzie, D.: The Pathogeny of Aural Cholesteatoma, J. Laryng. & Otol. **46**:163-189 (March) 1931.

16. Grove, W. E.: Primary Cholesteatoma of the Temporal Bone, Arch. Otolaryng. **10**: 398-408 (Oct.) 1929.

17. Teed, R. W.: Cholesteatoma Verum Tympani: Its Relationship to the First Epibranchial Placode, Arch. Otolaryng. **24**:455-474 (Oct.) 1936.

18. Lindsay, J. R.: Cholesteatoma Associated with an Isolated Perforation in Shrapnell's Membrane, Arch. Otolaryng. **20**:47-56 (July) 1934.

and Quittner [19] have all reported cases supporting the primary theory, basing their conclusions on the occurrence of cholesteatoma seemingly not associated with inflammatory reaction in the middle ear or mastoid. McKenzie,[15] Grove,[16] and, recently, Volk [20] have believed that all cholesteatomas are primary in origin and that suppuration follows after their extension into the tympanum or after perforation into the external canal. Many men [21] have provided ample evidence to show that congenital embryonic cell rests are extremely rare. Cholesteatoma is of very common occurrence. Holmes [13] reported that in 25 of his 303 cases cholesteatoma was found on the second operation, whereas none had been seen at the time of original operation. One is forced to conclude that while true cholesteatoma may occur as a primary tumor, such occurrence is rare, and some other explanation must be found for frequent findings of this tumor.

2. The invasion, or immigration, theory has for many years been the most widely held and is to be found in nearly all textbooks dealing with this subject. Politzer [22] reviewed carefully the theories on this subject and came to conclusions much the same as have those who are studying this problem today. This theory presumes that stratified squamous epithelium grows into the middle ear, attic, and antrum from a perforation of the tympanic membrane, particularly in the region of the pars flaccida. This perforation may occur as the result of middle-ear infection or may in some instances occur spontaneously. As later stated by Almour,[23] adhesive bands may form in the course of otitis neonatorum, closing off the tympanum. This results in a relative vacuum with sucking in of Shrapnell's membrane and eventual perforation in this area. Eggston and Wolff [6] demonstrated microscopically stratified squamous epithelium growing through a perforation into the middle ear and forming a cholesteatoma. Politzer [22] pointed out:

> The mere invasion of the epidermis of the external meatus into the tympanic cavity does not in itself lead to the formation of cholesteatomatous masses. This is shown by the epidermic blind pouches first described by Schwartze and also observed by the author in a vast number of specimens. These pouches extend from the external meatus into the cavum tympani and mastoid antrum, and may persist as such during the entire life of the patient. The inward growth of the epidermis of the external meatus towards the tympanic cavity, without the formation of cholesteatomata, takes place more often than is generally supposed. This is evident from many specimens of exhausted middle ear suppurations with extensive perforation of the membrane which the author has had occasion to examine, and in which no trace of an accumulation of epidermis in the tympanic cavity was seen macroscopically, but in which he could show microscopically in serial sections that the epidermis of the external meatus had extended to the inner tympanic wall. The invasion of the epidermis of the meatus leads to the secondary formation of cholesteatomata only when there is an excessive growth of epidermis in the external auditory canal, which is associated with a marked desquamation of the living membrane of the middle ear.

19. Friedman, M. D., and Quittner, S. S.: Cholesteatoma Verum of the Right Mastoid, Arch. Otolaryng. **28**:209-212 (Aug.) 1938.

20. Volk, B. M.: Primary Aural Cholesteatoma, Arch. Otolaryng. **53**:662-667 (June) 1951.

21. Dixon, O. J.: Otogenous Cholesteatoma, Ann. Otol. Rhin. & Laryng. **56**:986-998 (Dec.) 1947. Nager, F. R.: The Cholesteatoma of the Middle Ear: Its Etiology, Pathogenesis, Diagnosis and Therapy, ibid. **34**:1249-1258 (Dec.) 1925. Trumarkin.[11]

22. Politzer, A.: Diseases of the Ear for Students and Practitioners, Ed. 6, revised by M. J. Ballin, Philadelphia, Lea & Febiger, 1926, pp. 416-420.

23. Almour, R.: The Significance of the Squamous Epithelium in the Cause and Repair of Chronic Middle Ear Disease, Tr. Am. Acad. Opth. **35**:357-377, 1930.

The many authors [24] who have maintained this theory of ingrowth assume that the cholesteatomatous formations occur as a result of trapped epidermal elements which undergo profuse desquamation, most probably stimulated by the presence of inflammation or chronic suppuration.

3. The metaplasia theory was presented as early as 1873 by Wendt,[25] as cited by several authors. He believed that under the influence of chronic infection the epithelium of the middle ear would undergo a desquamative inflammatory reaction, thus producing cholesteatoma. A similar reaction has been observed in other regions of the body. Eggston and Wolff [6] stated that the mucous membrane of the middle ear is subject to cellular multiplication and metaplasia on relatively slight stimuli. Begley and associates [12] concluded that the metaplasia theory is the most plausible in the majority of cases and can explain the occurrence of cholesteatoma in any of its known circumstances.

In another recent paper, Begley and Williams [26] report two cases of cholesteatomatous cysts secondary to incomplete removal of cholesteatomatous matrix. They feel that the cholesteatomatous matrix may force itself into the Haversian canals of bone and persist in an eczematoid reaction, whereas a skin graft is less likely to do so. This was referred to earlier by Politzer,[22] who quoted Kirchner's findings of "cholesteatoma matrix extending into the Haversian canals of the bony wall surrounding the matrix" and felt that this explained the relapses following incomplete surgical removal of cholesteatoma. Begley and Williams [26] also state that "in certain instances it has seemed that the eventual result is better when skin grafting is not used, because of occasional tendencies in the grafted skin for an eczematoid reaction to develop."

COMMENT

My experience with these 11 cases of cholesteatoma formation following skin grafting of mastoid cavities leads me to question the views of Tumarkin,[11] who, as a result of his "quadruple flap" operation, was convinced that buried epithelium would not form cholesteatoma. All the evidence in the cases presented points to the formation of large masses of cholesteatomatous material from grafts of skin which have become overgrown by granulations. In all of the 52 cases in which operations have been performed since 1946, I have attempted to remove as completely as possible all traces of cholesteatoma and its matrix. This was accomplished much more completely than had been done prior to 1946. The masses of cholesteatomatous material appearing in these 11 cases have been far too extensive and have appeared in much too short a time following operation to have been the result of minute segments of matrix that might have escaped observation at opera-

24. Ballenger, W. L.; Ballenger, H. C., and Ballenger, J. J.: Diseases of the Nose, Throat, and Ear, Ed. 9, Philadelphia, Lea & Febiger, 1947, pp. 567, 676-680. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat, and Ear, Including Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1945, pp. 290-291. Kopetsky, S. J.: Surgery of the Ear, Ed. 2, New York, Thos. Nelson & Sons, 1947. Baron, S. H.: Modified Radical Mastoidectomy Preservation of the Cholesteatoma Matrix: A Method of Making a Flap in the Endaural Technic, Arch. Otolaryng. **49**:280-302 (March) 1949.

25. Wendt: Cited by Politzer [22] and by Eggston and Wolff.[6]

26. Begley, J. W., Jr., and Williams, H. L.: Cholesteatomatous Cysts Secondary to Incomplete Removal of the Cholesteatomatous Matrix. A. M. A. Arch. Otolaryng. **53**:147-152 (Feb.) 1951.

tion. I cannot but feel that these extensive growths of cholesteatoma have been the direct outcome of skin grafts which have become buried in granulation tissue. Perhaps they have been the result of an eczematoid reaction as mentioned by Begley and Williams,[26] or perhaps, just as occurs with invasion of epidermis into the tympanum or by metaplasia of the living epithelium of the middle ear, an inflammatory desquamative reaction is set up in the skin graft itself, producing a typical cholesteatoma formation.

It has been a source of disappointment to me to experience these unsatisfactory results with a procedure which has been so universally accepted. I am disturbed with the thought that a large group of surgeons who have enthusiastically followed this technique have not reported similar experiences. In a careful review of my recent operative and postoperative technique, the only factor that I can find to explain this unpleasant and dangerous complication is my employment of skin grafts. I feel fortunate that as yet none of these patients have required a secondary operation, but I cannot help wondering what might have happened if they had not been able to receive medical care.

SUMMARY

Eleven cases of cholesteatoma formation following the use of primary skin grafts in the radical mastoidectomy cavity are recorded.

A review of the literature concerning the histogenesis of cholesteatoma and a summary of the present opinions is presented.

The hypothesis is made that cholesteatoma may form as the result of an inflammatory desquamative reaction in primary skin grafts which have become overgrown by granulation tissue.

The question is raised as to the advisability of using primary skin grafts in the cavity following a radical mastoid operation.

Clinical Notes, New Instruments and Techniques

PLACE OF COLUMELLA IN SUBMUCOUS RESECTION

ALBERT P. SELTZER, M.D., Sc.D.
PHILADELPHIA

The columella is the only part of the nasal septum that is visible on the face. Its significance is not limited to the reconstruction of cosmetic surgery, since it is of particular importance in maintaining the cartilaginous septum in a median position, although it should always be given esthetic consideration.

In distortions and deformities of the nasal septum, whether congenital or resulting from injury caused by accident or disease, when a submucous resection is required to correct the septal fault the columella is always involved in the operation.

Two precautions are to be observed in relation to operations which include the columella. The first is to make sure that it will finally appear in the midline of the base of the nose, to insure symmetry between the two halves. The second is to avoid robbing it entirely of its normal mobility and thus to prevent giving it a fixity that interferes with the adaptation that is required for change in facial expression.

There are different methods of doing a restoration of the septum, although they vary chiefly in details. The widest variation from the customary is the Metzenbaum operation. This is not a resection, nor is it suitable in all conditions, but it is a replacement of the cartilaginous septum into the grooves which it normally occupies. In this technique the lower border of the cartilage is merely resettled in its relation to the columella.

In uncomplicated cases in which a submucous resection is carried out, the lower margin of the cartilaginous septum, or the cartilage graft which may be substituted for it, is sutured to the adjacent tissue of the columella without great difficulty.

The conditions will be quite different when there is not only deformity of the cartilaginous septum but also scar tissue which may have followed from accident or disease processes. When the scar tissue is abundant and dense, the requirements are entirely different. The columella itself may be involved, and a complete reconstruction will then be needed. To accomplish this, grafting will be required, particularly if the skin layer has been destroyed or has become thin and parchment-like from loss of its blood supply.

Many methods have been devised for use when such destruction has taken place. Grafts have been secured from a variety of sources, both from the patient's own tissues and from preserved materials. In all cases the skin graft must be as nearly as possible of the same texture and color as that of the original, and, necessarily, it must be hair-free.

Gillies (1949) has considered a variety of grafts which may suffice to present the requirements for this operation. These are not presented by him as original, since some of them were first used by several operators at about the same time as, and without the knowledge of, the others.

When there is complete loss of the columella, which may occur with saddle nose, a composite of skin and cartilage graft can be taken from the outer margin of the ear and used successfully if the blood supply to the columellar region is still adequate, but, since it is a free graft, it cannot be used if the surrounding tissue it atrophic. This method can be employed only as a second stage, unless the septum is in good condition for the attachment of the graft.

Depending on the state of the columellar tissue, flaps from the nearby surface of the face can be lifted and rotated; these may be taken from the nasolabial region on both sides, from the philtrum below, or from the lateral parts of the upper lip. The last can be used from both sides or from only one.

From the Department of Plastic and Reconstructive Surgery, St. Luke's Medical Center, Philadelphia.

Presented before the Pennsylvania Academy of Ophthalmology and Otolaryngology, Philadelphia, Jan. 16, 1952.

If a greater amount of tissue is required, a small nasolabial tubed pedicle can be made. This requires two stages, including, first, the preparation of the pedicled graft by suturing a thin fold of the skin with mattress sutures and, later, separation and rotation to the desired position for the columella.

An additional method, which gives good results when more superficial repair is indicated, is to raise a thin skin flap on either side of the basal surface of the nose and rotate it to the columellar position. Here the skin attachment which is left is obviously just beneath the nasal tip.

After a long nose with hanging tip has been reduced by submucous resection, Griesman (1950) reattaches the columella to the septum, using "orthopedic," or oblique, sutures. To be sure of satisfactory results, while the sutures are being tied the left hand is used to press the skin over the dorsum of the nose firmly against the septal cartilage and the right hand pushes the columella downward as far as possible toward the nasal spine, although the latter may already have been removed. If a buttress is needed to support the nasal tip, a graft is introduced within the soft tissue of the columella and placed so that the upper end is just below the tip of the nose while the lower end rests at a point corresponding to the junction of the lower and middle thirds of the columella. The graft is held in position by mattress sutures through the upper end of the columella.

When septal deformity causes obstruction of the nasal passages, making respiration inadequate, there may also be an abnormally wide columella, which should be narrowed to complete the reconstruction of the airway. This may be done by excising the superfluous tissue within the vestibule or by the removal of small triangular pieces of tissue at the lateral limits of the columellar base, with the incisions made to direct the apex of the triangle toward the cheek. The effect is not only to narrow the columellar base but also to draw the lower alar rim inward to form a labioalar junction which did not originally exist and in this way to produce a more esthetic curve of the nostril (Griesman, 1950).

Ersner (1949) presents a method for treating the columella after a radical resection, as in saddle nose, which provides it with support by attachments to the upper lateral cartilages. When this method does not suffice, a cartilage graft is inserted into the columella as a strut.

In a submucous resection, if insufficient cartilage has been allowed to remain along the dorsum and the lower extremity of the septal cartilage, there is danger of sufficient traction by scar tissue at the line of suture to the columella to draw the nasal tip downward enough to deform the nasal profile. To aid in avoiding this outcome, a method has been used which provides that the fresh edge of the septal cartilage be inserted in a shallow vertical incision of the columella. In this way, the cartilage lies between the two columellar walls and allows the free surfaces of the septal mucosa and of the columellar walls to be approximated on both sides. Two mattress sutures are then passed through the skin flaps of the columella and the septal cartilage, a straight needle with No. 000 absorbable surgical (gut) sutures being used. If this technique is not entirely suited to the case, the approximated edges of the mucosa and of the columellar skin may be joined together with No. 0000 absorbable surgical (gut) sutures, using a curved atraumatic needle. This method tends to avoid distortion from retraction by scar tissue; it also has the advantage of preserving the mobility of the lower septum, since the incisions into the columella are not deep enough to affect the entire connective tissue segment.

When, as sometimes occurs, there is distortion of the columella following unsuccessful surgery upon the septum, and the nasal tip has sagged, support can be supplied by replacing the columella with a tube graft made, preferably, from nearby tissues, choosing a location where there is good blood supply, as from near the base of the nose, from the sternomastoid region of the neck, or from the submental area. If there is a marked degree of sagging, a cartilage implant into the columella may be necessary. Joseph (1929) described the use of a flap from the upper lip, dissected so that the near extremity lies just at the labiocolumellar junction. He also used free grafts from the arm.

In a short presentation it is not possible to review all the details of the many variations of the operation on the columella which are associated with the basic technique of submucous resection. As in all surgery, the method used must be that one which most nearly meets the requirements of the existing conditions, and so it depends upon the choice and judgment of the individual operator.

2104 Spruce St.

CONTROL OF POST-TONSILLECTOMY PAIN

S. E. PENN, M.D.
PITTSBURGH

An operative procedure which is as frequently performed as is tonsillectomy may lead to an apathy on the part of the physician concerning the amelioration of the patient's morbidity. Postoperative pain and its sequelae (difficulty in swallowing, earache, etc.) are almost universal complaints of the patients. The rapid advances in anesthetic and surgical techniques have left this factor as one of the most distressing aspects of the entire procedure.

The approach to postoperative pain control has been largely through the use of salicylates and gargles. Application of an ice collar also has been suggested as affording some relief. The literature abounds with reports [1] of the salicylate-induced hemorrhage, and the use of gargles, while of dubious value, has been shown to interfere with the healing of the wound.[2] The local injection of procaine for pain control has resulted in some success,[3] but, unfortunately, the duration of activity is too short for this technique to be of real value.

The recent development [4] of "efocaine," a water-miscible, prolonged-acting, local anesthetic, appears to offer a sound approach to this problem. The mechanism of activity of this agent is based upon the pharmacologic fact that insoluble, slowly-absorbed materials exert an effect for a prolonged period. This is demonstrated by the use of the hormonal implants, suspensions of estrogens, and the suspensions of penicillin procaine. "Efocaine" consists of a solution of the normally water-insoluble anesthetic bases (procaine and butylaminobenzoate) in a nontoxic, water-miscible, organic solvent. This solution is at critical saturation limits; hence when it is diluted by even minimal quantities of aqueous fluid (extracellular fluid or serum) a complete deposition of the crystalline anesthetic agents occurs. This *in situ* crystallization creates an anesthetic depot which slowly and continuously releases the drug over an extended period.

Pharmacologic investigation [4] established the anesthetic duration of this drug to be greater than one week. A histopathologic study [5] demonstrated that there was no foreign body reaction, no neurodegeneration, no evidence of an inflammatory tissue reaction, and no encapsulation following its use. The solid anesthetic depot is completely absorbed from the site of injection. A particular study of the submucosal injection revealed this agent to be innocuous to this tissue.

Clinically, this drug has produced pronounced pain relief following major abdominal surgery [6] and anorectal surgery.[7] There were no untoward reactions observed in any of the patients studied, nor was there any interference with the process of wound repair.

From the Department of Otolaryngology, Montefiore Hospital.

1. Neivert, H.: Acetylsalicylic Acid and Bleeding from the Tonsillar Wound, Arch. Otolaryng. **47**:524, 1948. Livingston, G. S. and Neary, E. R.: The Question of Prothrombinopenic Hemorrhage from Post-Tonsillectomy Use of Chewing Gum Containing Acetylsalicylic Acid, ibid. **47**:1, 1948. Coombs, A. J.: A Study of Delayed Bleeding Following Removal of the Tonsils and Adenoids, Laryngoscope **60**:296, 1950. Jones, E. H.: The Prevention of Secondary Hemorrhage Following Tonsillectomy, South. M. J. **42**:124, 1949.

2. Cascales, M.: The Post-Tonsillectomy Phase, J. Med. **2**:630, 1948.

3. Debain and Lefèvre: Simplification of the After-Effects of Tonsillectomy in Adults by Local Anesthesia, Ann. oto-laryng. **66**:212, 1949.

4. Ansbro, F. P.; Iason, A. H.; Shaftel, H. E., and Bodell, B. B.: Efocaine, A New Concept in Prolonged Anesthesia, to be published.

5. Weinberg, T., Chief Pathologist, Sinai Hospital, Baltimore: Personal communication to the author.

6. Iason, A. H., and Shaftel, H. E.: A New Approach to the Problem of Postoperative Pain, Am. J. Surg. **83**:549, 1952.

7. Raicus, E.: A Critical Study of the Anesthetics Used in Ano-Rectal Surgery, M. Times, to be published. Gross, J. M.: Personal communication to the author.

In view of the desirability of such an agent to control post-tonsillectomy pain, it was decided to investigate its use for this purpose.

TECHNIQUE

In a consecutive series of 37 children and 8 adults "efocaine" was injected at the conclusion of surgery. General anesthesia was used for the children and local field block for the adults. Injections of 1 to 1.5 cc. of the solution were made into the anterior and posterior pillars on each side. The injections were made submucosally at various points about both pillars, care being taken not to deposit the solution too superficially. Since procaine is a potent vasodilator, the wound should be "dry," and all bleeding controlled prior to the start of the injection.

RESULTS

The results obtained in this series were most dramatic. A high degree of local pain control was achieved in every instance. There was no difficulty experienced in swallowing and none of the usual sequelae, such as earaches. The period of anesthesia lasted for approximately five to six days, after which some of the patients experienced a return of the local soreness. This was of a much lower order than is normally experienced and generally was of no concern. There were no untoward reactions and no interference with the healing of the wound, and there were no post-tonsillectomy hemorrhages observed. The patients were all greatly benefited subjectively, and convalescence was much more pleasant.

It should be noted that while the patient is apparently free of pain and able to swallow freely, the usual soft diet is to be strictly observed in order to avoid trauma to the wound. The surgeon should instruct the patient (or the parents) that, in spite of the absence of local distress, solid food is to be avoided for the usual length of time. While this series of 45 patients is admittedly not large, the uniform degree of success in the treated patients, in contrast to the universal complaints of the untreated controls, makes this most significant. This technique is worthy of wider application and will do much to reduce the morbidity of the patient following tonsillectomy.

SUMMARY

By injecting 1 to 1.5 cc. of "efocaine" into the anterior and posterior pillars following tonsillectomy, postoperative pain was controlled to a very high degree in a series of 45 patients.

Local anesthesia was observed for approximately five to six days. The patients were able to swallow freely and postoperative morbidity was greatly reduced.

There were no untoward results nor was there interference with wound healing observed in any of the treated patients.

3500 Fifth Ave.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D.
SHERMAN L. SHAPIRO, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

THERE has been quite a resurgence of German literature in the past year. The Scandinavian countries have kept up their pace, and the Italian is more and more in evidence. A great deal of the literature on rehabilitation now stems from acoustic physicists and teachers of the deaf and does not lend itself to abstracting. Such articles have been noted by mention and bibliographic reference. The American Medical Association issues a new list of approved hearing aids about every month, and it is our purpose to give the latest one before publication of this review.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF JULY 1, 1952

Audicon Models 400 & 415
Mfr., National Earphone Co., Inc.
20 Shipman St.
Newark 2, New Jersey

Audivox Model Super 67
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Symphonette Model

Beltone Mono-Pac Model M
Mfr., Beltone Hearing Aid Co.
1450 W. 19th St.
Chicago 8

Cleartone Model 500

Cleartone Model 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2
Mfr., The Dahlberg Company
2730 W. Lake St.
Minneapolis 16

Dysonic Model 1
Mfr., Dynamic Hearing Aids, Inc.
149 Church St.
New York 7

Electroear Model C
Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Model V-35

Gem Model V-60

Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Maico UE Atomeer

Maico Quiet Ear Models G & H

Maico Model J

Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal & Magnetic) Aurophone Model 200

1947-Mears Aurophone Model 98

Mfr., Mears Radio Hearing Device Corporation
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Model "Mercury"

Micronic Star Model

Mfr., Audivox, Inc.—Succesor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Audiomatic T-5

Microtone Classic Model T-9

Microtone Model T-10

Microtone Model T612

Microtone Model 45

Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506

Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Otarion Model E-4

Otarion Models F-1, F-2 & F-3

Otarion Models G-2 & G-3

Mfr., Otarion, Inc.
159 N. Dearborn St.
Chicago 1

Paravox Model D "Top-Twin-Tone"

Paravox Model J (Tiny-Myte)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC & YC-7) [Veri-Small]

Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radiocar Permo-Magnetic (Multipower)

Radiocar Permo-Magnetic (Uniphone)

Radiocar All-Magnetic Model 55

Radiocar Model 62 Starlet

Radiocar Model 72

Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mount Lebanon
Pittsburgh 16

Distr: Radiocar Corporation

Rochester Model R-1

Rochester Model R-2

Mfr., Rochester Acoustical Laboratories, Inc.
117 Fourth St., S. W.
Rochester, Minn.

Silvertone Model J-92

Silvertone Model P-15

Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck and Co.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99

Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Model 700

Sonotone Model 900

Sonotone Models 910 & 920

Sonotone Model 925

Sonotone Model 940

Sonotone Model 966

Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E

Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 500

Telex Model 1700

Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal

Tonemaster Cameo Model

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Model 95

Unex Midget Model 110

Unex Models 200 & 230

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2

Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 & 66

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Zenith Model 75

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex Semi-Portable

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid

Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50

Mfr., Sonotone Corporation
Elmsford, N. Y.

HEARING TESTS

Onchi [1] describes a modified tuning fork test that can be substituted for the audiometer. This is done by multiplying the damping constant of a tuning fork by the time difference between the hearing period of a deaf person and that of a normal person for the same tuning fork. With this test almost the same audiogram can be obtained as with the audiometer. The present tuning fork test in which the percentage scale of hearing is obtained depends upon the intensity of the blow used to vibrate the tuning fork. Since there is no standardized blow or a standard of intensity, we have a variable factor which will give variable results. A constant value is the time difference in hearing between that of the person with impaired hearing and that of one with normal hearing. The writer devised an acoustical instrument similar to a stethoscope. One ear piece is inserted into the patient's ear to be tested and the other into the examiner's ear. The fork is struck with a blow of random intensity. When the subject signals that the sound just becomes inaudible, the examiner immediately starts the stop watch and stops it when the sound becomes inaudible to himself. This gives the time difference of a tuning fork for the subject. The examiner should test his hearing acuity and that of a deaf person with an audiometer for each of the tuning fork intensities for which the damping constant of the tuning fork is to be measured. The damping constant of the tuning fork can be found in decibels per second by dividing the difference of hearing acuity between the two persons by the time difference in seconds. These damping-constant results compared very favorably with those obtained by an electroacoustical method. The examiner should determine his hearing acuity at various frequencies. To determine the true hearing loss of the subject, the hearing loss for the examiner is added to the hearing loss of the subject as shown by the modified tuning fork test, which is obtained by multiplying the damping constant of the fork by the time difference between the hearing period of the subject and of the examiner.

The author believes that this modified tuning fork test has several advantages. He says it is simpler and more durable for clinical use than the audiometer. The results can be compared with those of other hearing tests, such as voice and audiometry, because they are expressed in decibels. The time difference can be measured exactly and easily with this acoustical instrument, which he devised by moving the vibrating tuning fork from side to side in front of the instrument, producing a series of interrupted sounds like those of an audiometer. This test does not depend upon a standard stimulation of the tuning fork, like the previous tuning fork test. The fork can be sounded at any arbitrary intensity above the threshold intensity of the patient without altering the results of the test. The results can be plotted on the usual audiogram, and the curve will coincide quite accurately with the audiogram for the same patient. Onchi presents two examples in the paper.

Fowler [2] presents a brief history of the origin and use of the signs and symbols used in plotting threshold audiogram curves. He makes a plea and presents reasons for the use of simple uniform symbols which he devised. The use of the whole number scale has been recommended. However, those otologists still using the old

1. Onchi, Y.: Modified Tuning Fork Test That Can Be Substituted for the Audiometer, A. M. A. Arch. Otolaryng. **54**:693, 1951.

2. Fowler, E. P.: Signs, Emblems and Symbols of Choice in Plotting Threshold Audiograms, A. M. A. Arch. Otolaryng. **53**:129, 1951.

scale should not be inconvenienced, as it implies a change of only about 2.5 per cent. The dot is used to represent the right ear and the circle the left ear. If colors are used, it is suggested that red be used for the right and lemon or lead, or even blue, for the left. The bracket representing half of a box is the symbol used for bone conduction. The one opening to the right ([ ) is used for the right ear and the other, opening to the left ( ]), for the left ear. These fit nicely around the dot and circle without overlapping or touching each other when all have to be plotted around the same joint.

Fowler also discusses the problem of masking. He mentions the known fact that an air-conduction masking sound above 35 to 50 db., depending on the frequency, is loud enough to be sensed by the contralateral ear at its threshold or above if the bone conduction in the contralateral ear is not sufficiently depressed to prevent it. This diminution will occur whether the hearing in the tested ear is normal or decreased so long as its bone conduction is essentially normal. If the bone conduction in the ear being tested is reduced, then no crossed masking from the opposite side will occur unless the masking sound is loud enough to overcome the loss. There is no need of masking if the ear being masked has a greater loss by air and bone conduction than the ear under test, regardless of the degree or type of loss in that ear. However, these facts are not known to the otologists before the tests are made. Therefore, for true determinations of thresholds the masking sounds used in the contralateral ear must be kept below intensities detectable by the ear being tested. This can be determined by first testing without masking. If the hearing in the two ears differs by more than 35 to 50 db., masking should be used. It is not necessary to mask the poorer ear for air-conduction tests unless the bone conduction is normal or the loss less than that in the ear being tested. To determine the maximum nondetectable masking intensity which is to be applied to the better-hearing ear, the threshold for the test frequency is obtained and then the masking sound is introduced into the contralateral ear, its intensity being increased until it affects the threshold of the ear being tested. This value diminished by 5 db. represents the limit of intensity of the masking sound that can be used without altering true results.

A comparison of the "phon" scale for registering hearing loss (this is widely used in Europe, especially Germany) with the decibel scale is made by Lugli.[3] Whereas the decibel represents a ratio between audibility of a given sound and an initial or standard reference intensity of a sound whose value is 10^{-16} microwatts to the square centimeter at a frequency of 1000 cps, a phon is an expression of loudness using the decibel loudness of a tone of 1000 cps as the yardstick. While a phon and a decibel are the same at 1000 cps, the values begin to vary above and below that frequency. Curves are presented to show that in middle ear and conduction impairment the curves obtained by using the phon scale are more expressive of both this type of deafness and its extent than the decibel scale.

Using the Békésy audiometer, DeMaré and Rössler [4] experimented on masking in conduction deafness and inner-ear deafness. Audiograms are shown with pure

3. Lugli, G.: The Phon Scale Is Preferable to the Decibel Scale in the Graphic Registration of the Audiometric Examination for Clinical Purposes, Arch. ital. otol. **61**:601, 1950.

4. DeMaré, G., and Rössler, G.: Experiments in Masking in Conduction and Inner Ear Deafness, Acta oto-laryng. **38**:179, 1950.

tones as masking stimuli. In conduction deafness masking has less effect than in normal persons. This explains the relative improvement in speech intelligibility (paracusis Willissii). In nerve deafness the masking is the same as or greater than in normal persons. The role of auditory fatigue is also mentioned. The results are confirmed by the statements of 300 patients from their experience in noisy surroundings.

Kietz [5] examined the influence of an air-conducted masking tone on a bone-conducted signal of the same frequency. The complicated relations are discussed. A technic for the exact determination of the bone conduction has been developed.

Lewenfisz [6] describes tympanopneumatic and bone pneumatic tests based on experimental and clinical observations which showed that a pressure of 2 or 3 mm. of mercury in the external canal is sufficient for the immobilization of the stapes. A higher pressure immobilizes the round-window membrane and impairs both bone and air conduction. The mobility of the stapes as well as the function of the round window may be established by the method. It facilitates the diagnosis of otosclerosis.

Eby and Williams [7] substantiate the findings of Dix, Hallpike, and Hood, who found that the loudness recruitment phenomenon was present in patients with Ménière's disease and absent in patients with eighth nerve tumors and lesions at the cerebellopontine angle. This recruitment, however, did not apply to speech intelligibility, which was just reversed, being absent in persons with Ménière's disease and present in persons with neurofibroma, becoming more intelligible with increasing amplification up to a point. The writers tested 32 patients with unilateral deafness diagnosed as Ménière's disease and 1 patient thought to have a vascular accident involving the organ of Corti. Loudness recruitment was present in every patient, and in 17 of these evidence of overrecruitment was found. The same test was made on 16 patients with deafness due to degeneration of nerve fibers. In eight evidence of intracranial lesions involving the eighth nerve was found at operation; in all eight loudness recruitment was absent. In eight other patients, each with a probable lesion of the eighth nerve fibers, there was little or no evidence of loudness recruitment. Diplacusis binauralis dysharmonica was present in 23 of the 27 patients with end-organ deafness in whom this was tested and present in only 1 out of the 16 patients with nerve fiber deafness. Speech intelligibility tests were made in 17 cases of presumed lesions of the end-organ. In 15 cases speech intelligibility reached a maximum and then declined with further increases of intensity within the intensity range of the audiometer. In five of the six cases of nerve fiber degeneration, speech became more and more intelligible with increased intensity of sound until the limit of the audiometric scale was reached. There was also a wider span between speech reception threshold and most comfortable listening level in the cases of nerve fiber deafness as well as a greater tolerance or a higher threshold of discomfort for speech in these cases as compared with cases of end-organ deafness. Eby and Williams present these findings as further evidence indicating the presence

5. Kietz, H.: Masking in Bone Conduction Audiometry, Arch. Ohren- Nasen- u. Kehlkopfh. **157**:125, 1950.

6. Lewenfisz, H.: Tympano-Pneumatic and Bone-Pneumatic Tests, Otolaryng. polska **4**:17, 1950.

7. Eby, L. G., and Williams, H. L.: Recruitment of Loudness in the Differential Diagnosis of End-Organ and Nerve Fibre Deafness, Laryngoscope **61**:400, 1951.

of loudness recruitment, in contrast to the opposite results obtained of speech tests in their cases of nerve fiber deafness which seemed to indicate the absence of loudness recruitment.

The differential diagnosis by the Fowler loudness recruitment test of changes in the cochlear nerve, with reference to the loudness shock test, is described by Kobrak (London).[8] A high-pitched tuning fork struck at maximal intensity produces a shock phenomenon in the deaf ear. The patient may indicate that the sound was too shrill or make defensive movements with the head or hands. This may be used as a short orientating test for diagnostic differentiation between middle- and inner-ear lesions, as well as changes in the organ of Corti and retrocochlear changes. If the result is negative a more elaborate recruitment test must be made.

Groen [9] offers some advice on hearing aids made to order. In cases of recruitment automatic volume control of the hearing aid should be combined with an attenuation of 10 db. in the ear receiver. If there is increasing hearing loss for the higher frequencies, these frequencies should be "clipped." Stimulation of the degenerated part of the cochlea gives rise to disagreeable sensations without giving improvement in hearing.

The absolute evaluation of noise audiometry as a more accurate measurement of hearing loss is presented by Langenbeck.[10] Masking audiograms at various intensity levels of the special noise used are given for normal ears compared with ears with various types of impaired hearing. In pure conduction deafness the high level masking audiogram (80 to 100 db.) coincides with the normal ones. In certain cases of inner ear deafness (ganglion type) these curves are shifted to higher levels, whereas in other cases (hair-cell type) the normal picture is shown.

Three different types of inner ear deafness are considered by Kietz [11] in connection with the steepness of the pathological loudness curve. The author discusses some of the methods and results that have been published.

Lüscher [12] discusses the difference limen of intensity variations of pure tones and its diagnostic significance. He defined the difference limen for true intensity variations as the smallest change of tone intensity—namely, the smallest variation of the sound pressure—which is just perceived as a variation of the loudness. It can be expressed either in per cent or in decibels. The author prefers the use of per cent, which is a linear function and therefore clearer. The determination of the difference limen is a monaural method which may be applied in any form of deafness. In the normal it amounts to 10 to 16%. The relationship between per cent and decibels is as follows: Two per cent equals 0.18 db., 6% equals 0.54 db., 10% equals 0.92 db., 20% equals 1.94 db., and 50% equals 6.02 db.

8. Kobrak, F.: On Differential Diagnosis, by Fowler's "Loudness Recruitment" Test, of Changes in the Cochlear Nerve, with Reference to the "Loudness Shock Test," Confinia neurol. **10**:309, 1950.

9. Groen, J. J.: Hearing Aids Made to Order, Nederl. tijdschr. geneesk. **1**:158, 1951.

10. Langenbeck, B.: Noise Audiometry, the Absolute Evaluation, Arch. Ohren-, Nasen- u. Kehlkopfh. **158**:458, 1950.

11. Kietz, H.: Physical Features and Measurement of the Recruitment Phenomenon, Arch. Ohren-, Nasen- u. Kehlkopfh. **158**:471, 1950.

12. Lüscher, E.: The Difference Limen of Intensity Variations of Pure Tones and Its Diagnostic Significance, J. Laryng. & Otol. **65**:486, 1951.

The most suitable intensity for the determination of the difference limen is 40 db. above threshold, at which value there is little dependence on the frequency and the subject is easily able to notice the fluctuations in tones. When the degree of deafness does not permit an intensity of 40 db. above auditory threshold, the modulation can be carried out at a lower value without fundamentally altering the results. The determination is made by first making the regular hearing-loss curve. The difference limen is then determined at 40 db. above threshold. The greatest degree of accuracy is reached in the middle frequencies, from 250 to 4000 cps. It is less accurate below and above this area. The patient is also able to perceive the disappearance of the tone variations better than their reappearance. Therefore, the difference limen is considered as the tone intensity variation at which the loudness variation is just no longer perceptible. If the deafness is too pronounced to go 40 db. above threshold, the audiometer is set to the maximum intensity as 90 to 100 db. The technique requires a certain amount of experience and must be learned like any other type of audiometric examination. The patients learn the test rapidly and are fairly accurate in their responses. The margin of error is about 1% of the sound pressure variation as compared with maximal differences of the normal value—10 to 16% down to the maximum decrease of 2 to 6%. The results are represented by a vertical stroke in the curve of the threshold value at points corresponding to the frequencies used, with the percentages marked above the stroke.

Lüscher reports the results of investigations carried out in the Basel Otolaryngological Clinic and Polyclinic. Since this type of test is relatively new so far as its employment in various types of hearing impairments, the summary and conclusions of the writer are given in his own words.

The normal difference limen is dependent to a very great extent upon the tone intensity; it decreases rapidly as the tone intensity increases. At 10 decibels above threshold it amounts to 30 to 50 per cent, at 80 decibels to 2 to 4 per cent of the reference sound pressure.

A direct comparison of the difference limen and the recruitment phenomenon shows that the two phenomena are parallel and that the more pronounced the recruitment phenomenon is, the smaller is the difference limen. The difference limen is related to the recruitment phenomenon in two ways: (*a*) by the more rapid increase in loudness as such; (*b*) by the decrease of the difference limen as the tone intensity increases.

Distinction is made between a high difference limen (more than 16 to 20 per cent of the basic sound pressure), normal (8 to 16 per cent), moderately low (6 to 8 per cent) and very low (4 to 6 per cent). These values correspond to a tone intensity of 40 decibels above threshold and a hearing loss of 30 decibels or more in air conduction.

Conductive deafnesses due to alterations in the external meatus of middle ear, such as obstruction of the auditory meatus or of the eustachian tube, acute or chronic otitis media and otosclerosis with normal bone conduction have a normal difference limen.

Perceptive deafnesses are less uniform in their behaviour. The difference limen is usually diminished.

A marked decrease of the difference limen was in evidence in all the cases of acoustic trauma the author examined, and also in all patients with Ménière's disease.

On the other hand, the difference limen proved normal in five patients with retrolabyrinthine hearing disorders (one acoustic tumor, one cerebello-pontine tumor, one streptomycin damage, one parotitis epidemica, one central hearing disorder).

In cases of endogenous labyrinthine deafness, the difference limen is usually decreased, but normal values may be met with.

Patients with "concussion" of the labyrinth after severe head trauma are not uniform in their behaviour. Out of nine patients of this type, three had a decrease of the difference limen corresponding to the deafness, three had only a relative diminution at less perceived frequencies, and three had normal values.

Two patients with psychogenic hearing disorders exhibited a high difference limen of 20 to 40 per cent.

With rare exceptions, normal bone conduction is accompanied by a normal difference limen.

A reduction in bone conduction generally means a decrease of the difference limen. There are, however, three groups of exceptions: (1) diseases of the middle ear as an expression of conductive deafness with bad bone conduction; (2) in retrolabyrinthine hearing disorders, seldom in endogenous labyrinthine deafness, somewhat more frequently in "concussion" of the inner ear after severe head trauma; (3) in psychogenic deafness with a high difference limen.

Conductive deafnesses in the labyrinth as a consequence of changes in the labyrinth itself seem to be rare.

The decrease of the difference limen is not dependent on the frequency.

The greater the deafness is, the more rapidly the decrease in the difference limen occurs when the tone intensity increases, in so far as it is a deafness with a decrease in the difference limen, i. e. with recruitment phenomenon.

The difference limen and the recruitment phenomenon may be regarded from the topical diagnostic point of view as being equivalent.

The difference limen appears to be dependent on the function of the peripheral receptor, i. e. the sensory cells. An impairment of the difference limen indicates damage to the organ of Corti and thus allows peripheral disorders of the neural apparatus to be recognized. According to our experiences so far, the difference limen permits us to divide disorders of the neural apparatus into three groups: (1) disorders of the peripheral receptor, i. e. the sensory cells of the organ of Corti with decrease in the difference limen; (2) retrolabyrinth organic hearing disorders with normal difference limen; (3) psychogenic hearing disorders with a high difference limen. In so far as a retrolabyrinthine and psychogenic hearing disorder may be excluded, it permits a distinction to be made between conductive deafness and disorders of the neural apparatus.

Differential audiometry concerns itself with measurement of the faculty of discrimination of intensity, not the threshold of acuity for various frequencies, according to Lidén and Nilsson.[13] All frequencies are used at 5 to 80 db. above threshold. By these tests differential diagnosis may be made between those patients who show recruitment and those who do not. Patients with cochlear impairment are abnormally sensitive to changes in intensity once the threshold has been passed. The tests produce what the authors term a "diff-audiogram," and emphasis is put on the relation of this audiogram to acuity and recruitment.

Hakon Jorgenson [15] uses the headphones of the audiometer for the determination of bone conduction. The M. P. Peterson audiometer was used, which has rather large spherical head pieces as loud speakers, provided with insulating rubber rings. Tests were made on monoaurally deaf persons and on persons with normal hearing. If one ear was not deaf, it was tightly packed with paraffin-soaked cotton. Some were tested with one ear piece on the forehead, the other on the occiput. The author claims this method is accurate.

Zwislocki [16] suggests a better masking method for audiometry. After pointing out the shortcomings of present methods, he suggests use of a narrow frequency band composed of two bands on either side of the frequency being tested, which change in intensity with that of the testing tone. For masking in measuring bone

13. Lidén, G., and Nilsson, G.: Differential Audiometry, Acta. oto-laryng. **38**:521, 1950.

14. Footnote deleted.

15. Jorgenson, H.: Determination of Bone Conduction by the Headphone of the Audiometer, Acta. oto-laryng. **39**:16, 1951.

16. Zwislocki, V. J.: A Better Masking Method for Audiometry, Acta. oto-laryng. **39**:338, 1951.

conduction the masking sound must be proportionately decreased. This has worked out in practice, and the author asserts that an audiometer has been so constructed that it automatically adjusts the masking to the intensity and frequency of the testing tone.

The possibilities of the use of supersonic hearing in diagnosis is discussed by Zangemeister.[17] Patients with nerve deafness were tested at 34,000 cps. All except those who had labyrinth involvement, e. g., hydrops, were able to hear the sound. Normal persons sense supersonic sounds as a high note but not as a definite pitch.

A simple test for differentiating middle-ear deafness from inner-ear deafness is suggested by Miodonski.[18] The author claims that the distinction between inner-ear and middle-ear deafness can easily be made by conversation directed at the patient while he stops up both ears firmly with the fingers. In otosclerosis (good bone conduction) the change in hearing will be minimal. In inner-ear deafness (poor bone conduction) the patient will not hear at all. This test is offered as useful in busy outclinics.

Makita [19] studied the influence of postural changes on hearing. Possible changes in hearing with change of body posture were investigated in 30 persons with normal hearing by the Weber and Tomita tests, with the subjects lying on their sides. The majority showed lateralization of the Weber test while in this position.

Van Dishoeck and van Gool [20] used the bone-conduction vibrator of the audiometer to register the vibratory sense of the body surface for any frequency and intensity. The greatest sensitivity was found in the region between 200 and 300 cps. The vibratory sense is a means of communicating with the deaf. No hypersensitivity of the skin was found around the external ear canal in deaf patients; so it is suggested that other parts of the ear (tympanic membrane, basilar membrane, saccule) may serve. In some patients the vibrogram of the skin and the residual hearing in very deaf persons showed a remarkable resemblance.

G. E. Shambaugh Jr. and Carhart [21] report on the contributions that audiology has made to fenestration surgery, including a formula for the precise prediction of the hearing result. They stress certain important fundamental points and precautions to be noted in doing pure tone audiometry. First of all, the instrument must be in good working order and properly calibrated. This can be determined by testing 10 normal adult ears and taking the average threshold, which should be between 0 and 5 db. for each frequency. The testing technique used should be the ascending technique, testing from inaudibility to audibility. The bone-conduction audiogram can be just as reliable if certain additional precautions are taken. The bone-conduction unit must be checked often. This can be done by checking all bone-conduction audiograms, and when an obvious discrepancy is noted the calibration of the bone oscillator should be rechecked. This can be done by the otologist. Ten patients with perceptive losses of moderate degree are tested for both bone- and

17. Zangemeister, H. E.: Hearing of Ultrasound and Its Possibilities for Diagnostic Use, Hals-, Nasen- u. Ohrenarzt. **2**:387, 1951.

18. Miodonski, J.: An Inner-Middle Ear Test, Acta oto-laryng. **39**:296, 1951.

19. Makita, S.: Influence of Postural Changes on Hearing, Nippon Igaku Zasshi K. K. **22**:12, 1950.

20. Van Dishoeck, H. A. E., and van Gool, J.: The Continuous Vibrogram, Nederl. tijdschr. geneesk. **95**:467, 1951.

21. Shambaugh, G. E., Jr., and Carhart, R.: Contributions of Audiology to Fenestration Surgery, Arch. Otolaryng. **54**:699, 1951.

air-conduction sounds. The average loss for bone conduction and for air conduction should be equal for each frequency. If not, then a correction factor equal to the discrepancy should be applied to all bone-conduction audiograms made on the instrument. One should also remember that masking is required in all bone-conduction audiometry and a very quiet sound-proofed test room is more important for good bone-conduction audiometry than for air-conduction tests, as the untested ear is uncovered when bone-conduction tests are employed. The air-conduction curve of stapes ankylosis shows an early stiffness tilt with the tilt in the air-conduction curve downward toward the low tones. This becomes flatter as the ankylosis becomes more complete and becomes in effect part of the labyrinthine capsule, producing a greater loss for the high frequencies. The bone-conduction curve of stapes ankylosis shows a mechanical notching, which is largely reversed by fenestration. This notch, called the "Carhart notch," extends from 512 through 4096 cps, with its greatest depth at 2048 cps. It averages 5 db. for 512 cps, 10 db. for 1024 cps, 15 db. for 2048 cps, and 5 db. for 4096 cps. To determine the true cochlear reserve of a patient with otosclerosis, the bone-conduction curve is corrected by adding these respective figures to the bone-conduction audiogram: 5 db. to 512 cps; 10 db. to 1024 cps; 15 db. to 2048 cps, and 5 db. to 4096 cps. The air-conduction gap, which is the difference between the corrected bone-conduction curve and the air-conduction curve, measures the conductive component of the hearing loss. An air-bone conduction gap of less than 35 db. is a contraindication for operation. A patient with a maximum gap of 55 to 65 db. in addition to good cochlear reserve as manifested by a bone-conduction curve within 5 db. of the 0 line is the ideal patient. It has been shown that the fenestration operation leaves a "residue of unrestored conductive hearing loss" of 20 to 30 db. To predict the result of a successful fenestration operation, one should plot a curve 25 db. below the corrected bone-conduction curve. The method had been used in predicting the results in candidates for fenestration for a six month period before this paper was written. The results three months after operation in 75 patients were within 3 db. of the predicted results in 62.7% and within 9 db. in 88%. To compare and measure the performance of the fenestrated ear with the hearing-aid-aided ear, speech-reception-threshold determinations as well as discrimination scores were made before the operation and three to six months after the operation. These tests showed that in ideal cases surgery is the procedure of choice. In less suitable cases the results of surgery tend to be inferior to those obtained with the hearing aid in volume of hearing though superior in clarity.

Kos [22] examined the case records in 69 consecutive cases in which fenestration surgery was performed. He studied both the preoperative and the postoperative hearing status to determine whether some useful and simple but accurate audiometric method of selection could be used preoperatively to predict the results of the fenestration technique used. This analysis revealed that the bone-conduction threshold at 2048 cps and the cochlear reserve at 512 cps constitute elements of prognostic value at a rather high level of confidence. The characteristic tendency of the bone-conduction thresholds to form a descending curve from low to high frequencies naturally gives to the preoperative bone-conduction threshold of 2048 cps considerable weight in relation to prediction. The greater the loss at this

22. Kos, C. M.: A Statistical Study of Pure Tone Audiometry in Relation to the Fenestration Operation, A. M. A. Arch. Otolaryng. **54**:367, 1951.

frequency the more the acuity will be depressed at the two lower frequencies. The prediction formula formed in this study is: $X = 0.59x_2 - 0.14x_1 + 21.1$, in which X is the postoperative acuity, x_1 is the preoperative bone conduction at 2048 cps, x_2 is the cochlear reserve at 512 cps, and 21.1 is a constant which is the numerical differential between the mean values of the variables and the criterion audition. The numbers 0.59 and —0.14 are the relative weights found for the two variables. The cochlear reserve at 512 is the difference between the air and bone conduction at this frequency. The standard error of this estimate in this study proved to be ± 5 db. with 68% accuracy and ± 10 db. with 95% accuracy. This formula was tested in 25 instances, with 96% accuracy. The prediction equation derived is directly applicable only to the data obtained with the facilities and under conditions prevailing in the organization of the author. However, the method of arriving at such an equation may be applied to data obtained from other sources, and a comparable formula may be derived to aid in the selection and prediction of patients for fenestration surgery.

Marcus [23] employed encephalography as a diagnostic procedure for hearing and speech disorders in a group of 71 children. The majority, 42, were under 4 years of age. The tests were made under conditions of sleep, and the arousal response to stimuli such as the spoken voice, whistles, tom-toms, and cowbells, was used as an index of hearing and other sensory functions. Many of the children in this group showed other clinical abnormalities, as disorders of gait, slow development, behavior disturbances, and strabismus. Nineteen of the 53 children in whom a severe hearing loss had been established in the clinic showed electroencephalographic evidence of intracranial disorder. Nine of the 18 children having delayed, retarded, or unintelligible speech with some degree of hearing impairment also showed electroencephalographic evidence of intracranial disorder. All with an intracranial disorder showed in localization a preponderance in one or the other of the temporal lobes. The findings in these tests appeared to show that a disturbed cerebral control appears to be a factor in delayed, absent, or unintelligible speech in the young child and a possible factor in the clinical manifestation of deafness in the so-called congenitally deaf child. The test requires one to two hours, depending on the patient and whether sedation is necessary. In view of the difficulties and the formidable problems presented by hearing disorders in the very young, any additional test which can be of aid in making a diagnosis is valuable. Encephalography in sleep has been found valuable by Marcus in diagnosing hearing loss and speech defects in these children.

Saltzman and Ersner [24] report a group of six cases in which there were variations of the V-shaped and U-shaped audiograms. From their experience they believe, as do most otologists, that a high-tone V-shaped or U-shaped defect in the audiogram is most frequently caused by acoustic trauma. Infection is a less common etiologic factor. Low frequency defects of similar shapes are of obscure origin. The clinical picture presented by these cases suggests that the defects are either of congenital origin or due to an unrecognized attack of mumps or some other infection.

23. Marcus, R. E.: Hearing and Speech Problems in Children: Observations and Use of Electroencephalography, A. M. A. Arch. Otolaryng. **53**:134, 1951.

24. Saltzman, M., and Ersner, M. S.: The V-Shaped and U-Shaped Audiograms, Eye, Ear, Nose & Throat Month. **30**:247, 1951.

A comparative investigation of auditory fatigue tests was made by Griesen.[25] After analyzing Peyser's, Wilson's, and Theilgaard's auditory fatigue tests and obtaining conflicting results, the author concludes that none of them is reliable.

An entire supplement of the *Italian Archives of Otology, Rhinology and Laryngology* is devoted to a number of papers on "Problems of Audiology" contributed by the members of the Otologic clinic at the University of Milan.

An introduction by the head of the department, Prof. Luigi Pietrantoni,[26] describes the department of audiology, which is named in memory of Prof. F. Lasagna, a pioneer in this field. The department is modern in every way with soundproof room with a number of audiometers of different makes, including one "Elit," of Italian make, and a "Peters" audiometer for research. Charts are appended showing the various examination charts, which include audiograms graduated in both decibels and phons, recruitment charts, and various discrimination and intelligibility tests. A description of methods and technic is given. A close connection is maintained with the National Institute for Deaf Mutes as well as with acoustic physicists from the Milan Polytechnic School, the research department of the National Electro-Acoustic Institute, and Dr. Davelli, of "Elit," for the solution of technical problems.

The first paper in the issue deals with conduction deafness on the basis of personal investigations by Bocca and Pellegrini.[27] Their conclusion is that the average feature in this conduction is a flat hearing loss with a raising of the lower tones and a lowering of the upper tones. This is indicated not only by their investigations but by a number of physical and experimental data obtained by other workers. In addition to the air-conduction audiogram, a number of auxiliary tests are discussed and illustrated, such as bone-conduction, auditory-fatigue (greater in perception deafness), pain-threshold (less than normal in perception deafness), binaural loudness-balance and intensity-discrimination tests (done with the Peters research audiometer).

A contribution on presbycusis based on a study of 100 carefully chosen subjects between 50 and 90 years of age is offered by Agazzi and Vago.[28] They find that pure physiologic senile impairment is characterized by a progressive fall in air perception of high tones, by a correspondence between air and bone conduction, by a progressive lowering of the upper limit and a positive recruitment phenomenon. There is relatively little subjective disturbance experienced by the patient in proportion to the audiometric picture because the speech frequencies are only moderately involved. The authors consider the process a purely physicochemical one and consider pathologic changes which have been demonstrated by others in presbyopes as due to other causes; they also feel that diagnostic criteria should be evolved for differentiating pure senile impairment from that complicated by the presence of other conditions.

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25. Griesen, L.: Comparative Investigations of Different Auditory Fatigue Tests, Acta otolaryng. **39**:132, 1951.

26. Pietrantoni, L.: The Lasagna Audiology Department of the Otorhino-Laryngology Clinic of the University of Milan, Arch. ital. otol. (Supp. 5) **61**:1, 1950.

27. Bocca, E., and Pellegrini, A.: Conduction Deafness, Arch. ital. otol. (Supp. 5) **61**:8, 1950.

28. Agazzi, C., and Vago, A.: Presbyacusis: Clinical, and Audiometrical Considerations, Arch. ital. otol. (Supp. 5) **61**:28, 1950.

Azzi [29] presents an original contribution for a method of speech testing by means of what he calls logotomes. These are word fragments which he considers ideal for speech tests, since they constitute a stimulus only for the ear without involving the higher psychic centers. They are particularly suited as the Italian language is one of the few which has a constant correspondence between graphic signs and related phonetic sounds. The logatomes chosen represent a sequence of consonant-vowel-consonant-vowel and come in three groups: low logatomes, high logatomes, and mixed logatomes. The replies are graded according to the method of Egan into three different thresholds—threshold of detection, threshold of perception and threshold of intellection—and curves plotted. Live voice through a microphone circuit as well as tape recordings (which can be reproduced from a master record and distributed to other otologists) were used. In addition, short sentences for determining social adjustment as well as for the Doerfler-Stewart test for malingering and psychogenic deafness are also included.

Bocca and Pellegrini [30] discuss the possibility of calculating the loss for speech perception on the basis of the pure-tone threshold. They sum up the work of Fletcher and the contributions of Fowler which are not applicable to Italian-speaking persons. Their conclusion is that only actual speech tests give precise indications for judging the auditory function in a social environment. This test should be made exclusively by means of the registered voice or the monitored live voice. If the essential apparatus is not available, it is better to calculate the speech loss from the weighted losses for the four speech frequencies than to resort to the old method of speech testing. For this purpose the authors propose a modified scale for speech frequencies but stress that loss should be calculated in decibels rather than in percentages. They caution not to rely on the method in perceptive deafness, in which speech perception is extremely variable in comparison with hearing for pure tones.

Another article by Bocca and Pellegrini [31] in the same issue offers a statistical study on the phonetic composition of the Italian language based upon data obtained from the analysis of 1,500 words contained in written specimens of speech on the most various subjects. On the basis of these results they have composed six different lists of 50 bisyllabic words which represent faithfully the phonetic composition of Italian and which have proved to be satisfactory from the standpoint of homogeneity and intelligibility. They recommend widespread national use of their lists as a means of obtaining reliable data on speech reception thresholds in pathologic ears and urge that adequate electroacoustic equipment and careful determination of normal thresholds be employed.

Bocca and Camisasca [32] contribute a study of what they term the inertia of the hearing apparatus. This is a complex phenomenon involving the mechanical inertia of the sound-transmitting apparatus, persistence of neuroacoustic excitation, and

29. Azzi, A.: Vocal Acumetry Tests in Italian, Arch. ital. otol. (Supp. 5) **61**:45, 1950.

30. Bocca, E., and Pellegrini, A.: The Possibility of Calculating the Loss of Speech Perception on the Basis of the Pure Tone Threshold, Arch. ital. otol. (Supp. 5) **61**:103, 1950.

31. Bocca, E., and Pellegrini, A.: A Statistical Study on the Phonetic Composition of the Italian Language and Its Practical Application to Speech Auditometry, Arch. ital. otol. (Supp. 5) **61**:116, 1950.

32. Bocca, E., and Camisasca, L.: Studies on the Inertia of the Hearing Apparatus, Arch. ital. otol. (Supp. 5) **61**:142, 1950.

auditory fatigue. They consider auditory inertia an important factor in determining the behavior of the normal ear in the presence of interrupted sounds of high intensity. The phenomenon was determined by observing the masking effect of interrupted sounds or noises on the plain sound and on the voice. Both the masking and the masked sound were delivered from an audiometer through separate receivers in a free field, the experiment involving masking sounds repeated at different time intervals. The authors' findings indicated that the masking effect of the interrupted sound varies owing to the length of interruption, the intensity of the masked and masking sounds and the frequency of the masked sound. A sound of sufficient intensity to mask another pure sound of a certain intensity continues its effect when the interruption does not go below 120 milliseconds. For speech the masking effect of an interrupted sound is most evident with interruptions lasting 250 milliseconds.

Two papers dealing with paracusis Willisi are included in the symposium. In the first one, Azzi and Invernezzi [33] discuss the various theories regarding this phenomenon and present a new one. Having conducted tests on the masking effect of a noise of a certain intensity on pure tones for which the threshold had previously been determined in various types of deafness, they obtained different curves for each type of deafness. The exploitation of these data is proposed for determining the differential diagnosis between perception and conduction deafness and also serves as the basis of a new hypothesis to explain the fact that paracusis Willisi can be found in otosclerosis but not in adhesive middle ear processes.

Another paper deals with the objective determination of paracusis willisiana. Ettore Bocca [34] indicates that, contrary to the reports of some previous investigators, lowering of the threshold by air conduction for pure tones or speech in the presence of noise cannot be utilized as an objective test for this condition, although it is undeniable that the masking effect of noise is less marked in otosclerotics than in normal-hearing people. His experiments indicate, however, that bone masking of low frequency and high intensity as performed with an Edelmann fork of 100 cps at an intensity of approximately 80 db. fails to cause any change of threshold for air-borne sounds or tones in otosclerotic patients, whereas a change of threshold of varying degree (10 to 25 db. above the original figure) is demonstrable in persons with nonotosclerotic conduction deafness and in normal-hearing persons. Experiments were conducted by the author on 170 subjects, including 70 with otosclerosis, and results in the latter group have conformed to this fact in every case. Paracusis willisiana is explained by the fact that in persons with otosclerosis no masking by bone or air occurs as a result of surrounding noise, whereas bone masking takes place in persons with nonotosclerotic-conduction deafness and both bone and air masking occur in normal persons. The fact that sounds of equal loudness and frequency when transmitted by air cause a constant masking effect on a similar tone or sound even in persons with otosclerosis (though less than in normal persons) suggests that bone conduction is the factor which accounts for paracusis willisiana. The authors suggest that in otosclerosis bone-conducted vibrations win the initial resistance of the transmission apparatus to displacement, thus making it possible

33. Azzi, A., and Invernezzi, M.: The Derivation of the Threshold and the Use of Masking for Diagnosis in the Artificial Reproduction of Paracusis Willisii, Arch. ital. otol. (Supp. 5) **61**:154, 1950.

34. Bocca, E.: A New Method for the Objective Demonstration of Paracusis Willisii, Arch. ital. otol. (Supp. 5) **61**:181, 1950.

for weaker stimuli to pass through. They urge the bone-masking test as a simple and rapid diagnostic test for otosclerosis and state that in their hands it has been in agreement with the results of the Gellé test and with other clinical data.

The theoretical and practical effects of loads placed in the oval and round windows were studied by Staffieri.[35] After reviewing the publications of other writers on the subject with special reference to the work of Hughson and Pohlman, he reports the results of his own experiments on nine patients. He concludes that it is possible by this means to obtain an improvement of acoustic function if the windows of the internal ear have not been blocked by cicatricial tissue. Placing a cotton wisp dipped in petrolatum near the round window gives an increase in perception for low and medium frequencies; placing a similar wisp near the oval window results in a pantonal improvement, with the increase most marked for medium and high frequencies. When the material is placed near the oval window, there is in some instances a widening of the audiometric field, with a displacement of the bend either to the left or to the right. Bone conduction is improved in some patients regardless of whether the material is placed in the oval or in the round window. In discussing the mechanism by which the improvement is brought about in these patients Staffieri considers the various prevailing theories but considers it most likely that the improvement results from establishment of favorable conditions for the sound carriers to transmit impressions to the endolabyrinthine fluids.

A short paper by Bocca and Pellegrini[36] deals with estimates of hearing for speech for medicolegal purposes. The authors point out that there are a number of different methods of evaluating speech loss in terms of percentage loss for pure tones and that these methods fail to give an accurate idea of the practical loss of hearing for speech. They suggest that in medicolegal cases it would be useful to divide deafness into four degrees: first degree, 25 to 34 db.; second degree, 35 to 60 db.; third degree, 60 to 100 db., and fourth degree, above 100 db. Such a division, they believe, would be simpler from a medicolegal standpoint than an attempt to use a percentage basis.

Villata[37] comments on tests for simulation which can be done with the audiometer. Four are given: (*a*) repeated pure tone tests taken hours or days apart; (*b*) the Lombard test; (*c*) the Stenger tests; and (*d*) the Doerfler-Stewart test. The last, developed during the war in this country, involves feeding to the patient, who is sitting in a soundproof room, speech against a background of noise transmitted from another room through a loud-speaker. In persons with organic deafness or in persons with normal ears the speech can be distinguished even if the background noise is 10 to 15 db. higher than the intensity of the speech which is being transmitted. Simulators will refuse to repeat the words long before the level of the background noise reaches the intensity level of the speech. Of all the tests, the author considers the one involving repeated tests and the Stenger to be the most reliable. The Stenger test can be done with a double receiver. The Doerfler-Stewart

35 Staffieri, M.: Experimental Tests in Man on the Functional Effect of Loading the Windows of the Labyrinth, Arch. ital. otol. (supp. 5) **61**:195, 1950.

36. Bocca, E., and Pellegrini, A.: Evaluation of the Hearing for Speech in the Medico-Legal Field, Arch. ital. otol. (supp. 5) **61**:218, 1950.

37. Villata, I.: Modern Simulation Tests and a Critical Evaluation of Results, Arch. ital. laring. **62**:20, 1951

test, while constituting a useful test for bilateral deafness, does not distinguish between simulation and functional deafness.

A study of discrimination for intensity in bone conduction was made by Pirodda.[38] Fifteen subjects, 3 normal and 12 with various hearing defects, were tested. The researches of Békésy, Lüscher, Hallpike, Fowler, and others were reviewed. The ear not under examination was excluded by masking. The results showed that for both normal persons and persons with hearing defects there is complete agreement between the findings for air conduction and bone conduction. This fact is taken by the author to indicate that the recruitment phenomenon is completely independent of the middle ear. He considers also that bone conduction is just as suitable as air conduction for testing recruitment.

Caliceti [39] discusses the measurement of tinnitus, which he calls acufenometry. This is done by balancing various audiometric frequencies and intensities against the patient's subjective sound until the proper band and intensity are identified by the patient. The test is done in the opposite ear. He quotes Fowler to the effect that if the tinnitus can be masked easily by throwing the appropriate audiometric sound into either the affected or the opposite ear, a cochlear origin is indicated; on the other hand, if the tinnitus can be masked with difficulty or not at all, it is of central origin. The procedure is also of value in demonstrating simulation of tinnitus, since the malingerer cannot reproduce the appropriate frequency and intensity on different examinations. The author has adopted a system of symbols for indicating the frequency of the tinnitus as well as the ear involved which can be shown on the same chart as the audiogram.

Speech audiometry in the assessment of deafness is described by Dickson and Chadwick.[40] Equipment, test routine, and calibration of the speech tests used in the Central Medical Establishment, Royal Air Force, are demonstrated. Speech audiograms of persons with various types of deafness and of persons wearing hearing aids are shown. In fenestrated otosclerotic persons the average gain for speech was about 14 db. and for pure tones (512, 1024, and 2048 cps) about 9 db. The authors state that speech audiometry will give a truer assessment of improvement obtained by surgery or hearing aid than will pure tone audiometry.

Benson, Davis, and several others [41] sent a letter to the editor of *The Laryngoscope* telling him of two new recorded spondaic word tests that resemble the familiar auditory tests No. 9 and No. 14 of the Psycho-Acoustic Laboratory of Harvard University. These were prepared by the Central Institute for the Deaf with the collaboration of the Technisonic Studios. These new tests differ from the No. 9 and No. 14 tests in that the vocabulary is confined to very familiar words suitable for children as well as adults, and also the pace is faster and more suitable for talk-back instead of write-down responses. These tests are called W-1 and W-2, and the material used is a list of 36 spondaic words selected from the larger group in auditory tests Nos. 9 and 14. Six different scramblings of the same list of 36 words have been recorded on discs. The auditory test W-1 is recorded at a constant intensity and

38. Pirodda, E.: Behavior of the Threshold of Discrimination for Intensity in Bone Conduction, Oto-rino-laryng. ital. **19**:152, 1950.

39. Caliceti, G.: Technique of Acufenometry, Oto-rino-laring. ital. **19**:140, 1950.

40. Dickson, E. D. D., and Chadwick, D. L.: Speech Audiometry in the Assessment of Deafness, J. Laryng. & Otol. **64**:464, 1950.

41. C. I. D. Auditory Tests W-1 and W-2, Letter to the Editor, Laryngoscope **61**:838, 1951.

is designed for use with a step attenuator to obtain the greatest possible flexibility and speed in finding thresholds for speech. The W-2 auditory test is a descending-level test. Each operator must establish his own normal values for each type of test with his own equipment and under his own particular acoustic conditions. However, if the recommendations concerning apparatus that are given in the manual that accompanies the new readings are followed, normal threshold values should not differ from those of the authors by more than 2 to 3 db. These recordings are available through the Technisonic Studios. The complete list, prices, and address of the studios is given in the letter. It is hoped that various laboratories and clinics interested in the standardization of speech audiometry will try these new recordings and communicate their criticisms and suggestions.

After five years of practical experience with the various articulation tests, Eldert and Davis [42] have found that the Rush Hughes recordings of eight phonetically balanced (PB) word lists, made at the Central Institute for the Deaf in 1945 and used since then, have certain shortcomings. They found that the wide range of difficulty among individual test items is uneven and not ideal. There is an irregularity at the 20 db. level in the shape of the average articulation functions for all lists. This depression is significant for the pure conductive and for the conductive mixed groups and seems to be a spurious characteristic of the Rush Hughes recordings. The reliability of the Rush Hughes test is not very high. At levels 40 to 55 db. above the threshold for Auditory Test No. 9 the standard deviations around the mean scores never become much lower than 6%. The writers hope that these findings and experience will prove valuable to those who attempt to produce better recorded versions of the PB lists in the future.

Saltzman and Ersner [43] present some psychophysiologic aspects of speech hearing. The brain integrates speech sounds into meaningful patterns. The cerebral cortex has the function of filling in gaps to complete a word picture. Loudness perception and frequency discrimination are functions of the cochlea. In addition, the cochlea has the function of transmitting speech sounds with adequate characteristics to enable the brain to formulate meaningful patterns. This factor may be designated the "transmission-fidelity factor." When this is interfered with, amplification of speech sounds will be of little or no aid toward intelligibility. This condition exists in Ménière's disease with a marked low frequency loss. This deficiency in the transmission-fidelity factor may be partial or total. In otosclerosis this factor is largely influenced by sound power. The normal ear is capable of hearing certain weak consonants at a greater distance than other consonants with much more sound power. This advantage is lost to a variable degree in degeneration of the inner ear, especially in presbycusis with high-tone deafness. These considerations may help to explain why certain patients are benefited more by hearing aids than others.

The question of avoiding deafness in tuberculous meningitis treated with streptomycin is considered by Kluyskens.[44] The author claims that if the administration of

42. Eldert, E., and Davis, H.: The Articulation Function of Patients with Conductive Deafness, Laryngoscope **61**:891, 1951.

43. Saltzman, M., and Ersner, M. S.: Psychophysiology of Speech Hearing, A. M. A. Arch. Otolaryng. **53**:182, 1951.

44. Kluyskens, P.: Is Deafness Avoidable in the Treatment of TB. Meningitis with Streptomycin Belg. tijdschr. geneesk. **6**:1015, 1950.

streptomycin is stopped as soon as the patient shows some loss of hearing for high tones, no further damage to the hearing occurs. Once the speech frequencies are affected, total loss of hearing is unavoidable.

CONGENITAL AND OTHER TYPES OF DEAFNESS

Kinney [45] tried to clarify in his own mind some of the pertinent questions pertaining to hereditary deafness. A complete survey of the literature showed first a marked confusion in the use of descriptive terms concerning deafness and secondly, a great paucity of pathological material. The writer gives his own classification based on etiology and pathology.

Congenital deafness
- I. Hereditary
  - A. True Mendelian
    - 1. Dominant
    - 2. Recessive
  - B. Erythroblastosis fetalis
  - C. Secondary to other traits
- II. Malformations of external or middle ear
- III. Occurrences in mother during first trimester of pregnancy
  - A. Rubella or other virus diseases
  - B. Toxic poisoning—quinine, alcohol
- IV. Intrauterine accidents
- V. Birth injuries

All cases of hereditary deafness are congenital, but not all cases of congenital deafness are hereditary. The writer finds this question is far from solution and feels that pathologic study of uniformly processed specimens is very important, as is experimental study as to the possible alteration in the transmission of hereditary deafness.

Goodhill [46] studied two groups of patients with infantile perceptive deafness with reference to etiologic factors. One group was examined otologically and audiometrically, and a larger group was analyzed by means of answers to a questionnaire. Of the 904 patients studied by means of the questionnaire, 186, or 20.5%, had deafness due to maternal rubella in the first three months of pregnancy. Nineteen patients were examined in detail otologically. The audiometric pattern was almost always a purely perceptive loss. The scattergram shows a rather widespread distribution of the threshold levels. The majority of the curves were in the 40 to 70 db. range. The percentage of cases of deafness due to maternal rubella reported in the questionnaire may be relatively high, as these cases represent a large number of children who were conceived during a known rubella epidemic in this country. This disease plays a striking role as an apparent cause of deafness and should evoke important public health recognition. Questions of intentional exposure of unmarried girls, use of preventive and therapeutic sera, and therapeutic abortion merit the serious consideration of the entire medical profession. Goodhill

45. Kinney, C. E.: The Pathology of Hereditary Deafness, Ann. Otol. Rhin. & Laryng. **59**:1117, 1950.

46. Goodhill, V.: The Nerve Deaf Child: Significance of Rh, Maternal Rubella and Other Etiologic Factors, Ann. Otol. Rhin. & Laryng. **59**:1123, 1950.

also produced further evidence to add erythroblastosis due to Rh-factor incompatibility to the list of etiologic factors responsible for infant nerve deafness. He reports two new supporting cases.

Hiller [47] investigated 42 cases of congenital deafness, of which 32 were due to rubella occurring in the mother in early pregnancy and 10 were not due to rubella. The average degree of deafness was less in the cases due to rubella. Audiograms were of diverse patterns. In general, low tones were less affected than high, but the downward trend of the curve less steep than usual in inner ear deafness. The incidence and severity of the deafness and of the accompanying rubella retinitis appeared dependent upon the severity of the maternal infection. Congenital deafness with the typical curve of inner-ear deafness is probably hereditary. Atypical curves may be the result of prenatal infection, such as maternal rubella.

Packer [48] studied the influence of maternal measles on the unborn child. Eighteen cases of maternal measles during pregnancy were traced. Twelve offspring had no defects. The measles in this group occurred between the 1st and the 31st gestational week. Four pregnancies terminated prematurely. One child was Mongoloid; the mother had measles in the fifth week of pregnancy. One child was deaf; the mother had measles in the eighth week of pregnancy.

Congenital deafness, pigmentary degeneration of the retina, and amentia appear in the histories of three sisters, described by Peterson.[49] The literature on the subject is reviewed, and the etiology and hereditary factors are discussed. Early educational training is advised in all cases of deaf-mutism unless there is also primary amentia.

Defective hearing after meningococcal meningitis was studied statistically by Trolle.[50] The literature before and after the introduction of chemotherapy and antibiotics is reviewed. The percentage of patients found with hearing defects varies from 50 to 2.5% in different series of cases. The pathology is described as infiltration of the eighth nerve with purulent exudate, secondary descending degeneration of the cochlear nerve structures, purulent labyrinthitis, septic embolism in the internal auditory artery, labyrinthine hydrops with tissue lesions in the internal ear, and cerebral disturbances. Otitis media plays a small part only. Three hundred twenty-seven survivors over the period 1920-1945, inclusive, were studied, giving results before and with chemotherapy. The mortality was reduced from 68.8 to 11.9%. The incidence of hearing defects was also greatly reduced in spite of the greater number of survivors. Between 1926 and 1929 intramuscular administration of quinine was used with a high percentage of hearing defects in the survivors. Examples of audiometric charts of the different hearing defects are shown.

Fowler [51] presents pertinent data from 26 cases of sudden deafness to determine a possible common etiologic factor. Seventy-seven per cent of the patients were under 40 years of age. There was monaural deafness in 20 and binaural deafness

47. Hiller, B. H.: Rubella Congenital Inner-Ear Deafness, J. Laryng. & Otol. **64**:399, 1950.

48. Packer, A. D.: The Influence of Maternal Measles on the Unborn Child, M. J. Australia **1**:835, 1950.

49. Peterson, B. H.: Congenital Deafmutism: Pigmentary Degeneration of the Retina and Amentia, M. J. Acoustics **2**:854, 1950.

50. Trolle, E.: Defective Hearing After Meningococcal Meningitis, Acta oto-laryng. **38**:384, 1950.

51. Fowler, E. P.: Sudden Deafness, Ann Otol. Rhin. & Laryng. **59**:980, 1950.

in 6. All except three gave a history of emotional episodes preceding the sudden deafness. Of the 30 totally deaf ears, tinnitus was present in 10. In 14 patients tinnitus was present in the affected ear preceding the deafness. Vertigo was present either before, during, or after the sudden deafness in 21. In all these cases, including those reported by other writers, there seems to have been a neuritis or degeneration of the eighth nerve of unknown origin. It is possible that this was caused by an anoxia, ischemia, and altered metabolism. Fowler suggests that other parts of the body be searched for evidences of vascular spasm which could affect the inner ear. Sludging of the blood is one condition which is closely associated with vascular spasms. This phenomenon can be observed in the conjunctival vessels. In five cases of this series, in which the patients were examined for this phenomenon, it was distinctly evident. The others were not examined early enough. Fowler believes that it is probable that the neural irritations and degenerations causing sudden deafness, tinnitus, and vertigo were caused, at least in part, by the sludging phenomenon and the emotional episodes preceding the onset. These episodes could affect the neurovascular nervous mechanisms through autonomic nervous system response. Sludging of blood occurs in a large variety of conditions and can even be produced by certain hormones, epinephrine, estrogen, and emotional upsets. This sludging could probably cause deafness when there is some hereditary anatomical condition or previous disease or injury which favors sludge sticking in the small terminal vessels of the cochlea. A transient sludge could account for Ménière-like attacks, a transient dizziness and deafness. A sludge remaining for longer periods might produce sudden deafness. Fowler suggests that in cases of sudden acute deafness and blood sludging, antisludging therapy be used, such as injection of 500 cc. of 0.1% procaine hydrochloride solution intravenously.

Blegvad [52] calls attention to the large number of cases of deafness in World War I following explosion or shock or both. Many patients recovered spontaneously and soon. The patients transferred to hospitals at home did not do so well. The diagnoses were "concussion of the labyrinth," "acoustic deafness," "labyrinthine hemorrhage," "destruction of the organ of Corti," etc. In many cases hearing suddenly returned after psychic shock. The same also was true of hardness of hearing. By World War II audiometry had been introduced and showed many of these patients to have a loss at 4000 cps, in some extending to 8000 or even 12,000 cps—that is, it resembled "boilermaker's deafness," which is accepted as due to organic changes. Dr. Truex reported from Deshon Rehabilitation Center that it was not until 1945, when patients were referred for psychiatric examination and the diagnosis of psychogenic deafness was made more efficiently, that nearly twice as many such were discovered as before. Many recovered hearing for spoken or whispered voice, but the dip in high tones remained. In some patients an organic lesion remained, but the deafness for the human voice was a "psychogenic overlay." If this overlay was removed, the hearing became normal for speech. The necessary frequencies for hearing voice are 500 to 2000 cps.

The symptoms suggestive of psychogenic deafness are listed: e. g., agitation and anxiety during the interview; variation in the functional acoustic tests on different days; discrepancy in the speech and pure tone audiometry. The Doerfler-Stewart

52. Blegvad, N. R.: Copenhagen: Psychogenic Deafness (Emotional Deafness), J. Laryng. & Otol. **65**:166, 1951.

test patient hears speech over a gradually introduced noise until it reaches a level with or 10 to 15 db. over the speech level.

When the patients seem to lip read too well, various other tests are described and methods of treatment are given.

Seelenfreund [53] presents a new method of diagnosis of deafness due to circulatory disorders. He tests the hearing and retests on an inclined board, head down, measuring the change of hearing due to change of blood supply, which may suggest the use or non-use of vasodilators.

Pokorny and Hohlbrugger [54] report a case of transitory central disorder of hearing. A 65-year-old pianist with absolute pitch suddenly suffered a lowering of all tones in the region of d-3 and d-4. In the same region the octaves were heard as ninths. The patient was found to have a perception deafness of about 60 db. for frequencies above 3000 cps. He soon adapted himself to his erroneous perception. Treatment was otherwise unsuccessful.

Harbert and Sataloff [55] discuss functional, or nonorganic, deafness. They define it as a condition in which the patient does not fully utilize his residual hearing as determined by numerous available tests for detecting organic loss in auditory acuity. This condition is discussed under the following headings: psychological; malingering, either transient or due to a personality disorder; psychoneurotic, which is subdivided into anxiety neurosis as neurasthenia, conversion hysteria, and hypochondria. Some of the characteristic features of each of these is presented. Some of the findings which should alert the otologist to the possible presence of a functional hearing loss are the following: a medical history which could not possibly explain the patient's condition, such as a profound hearing loss following the instillation of ear drops into the canal of the ear; either no improvement at all with a hearing aid unless the patient is totally deaf or too great an improvement with an aid, especially when the controls are set at minimal amplification; marked fluctuation in hearing acuity as determined by any single test; inconsistency in the results of two or more tests; psychiatric manifestations, such as emotional symptoms or their vegetative or somatic signs; reflex reactions produced by sound as cochleopalpebral reflexes or psychogalvanic skin resistance test. A brief outline to the therapeutic approach is also given.

Lobel [56] reports further on the use of vitamin A in injectible form in the treatment of impaired hearing. Although the hearing loss was not influenced, in some instances the accompanying tinnitus was greatly relieved. Lobel believes, as do Mellanby and other investigators, that deafness is a nutritional deficiency disease. A significant finding in patients with deafness with or without tinnitus not due to systemic disease was a low level of vitamin A. When this was associated with the finding of a high cholesterol level as well as increased pyruvic acid, the administration of vitamin A and vitamin B complex produced a very rapid increase

53. Seelenfreund, B.: A New Method of Diagnosis of Deafness Due to Circulatory Disorders, Acta oto-laryng. **39**:238, 1951.

54. Pokorny, G., and Hohlbrugger, H.: Transitory Central Disorder of Hearing: Case Report, Hals-, Nasen- u. Ohrenarzt. **2**:59, 1950.

55. Harbert, E., and Sataloff, J.: Functional Deafness, A. M. A. Arch. Otolaryng. **54**:38, 1951.

56. Lobel, M. J.: Is Hearing Loss Due to Nutritional Deficiency? A. M. A. Arch. Otolaryng. **53**:515, 1951.

in hearing levels. In 30 per cent of the patients under study with normal blood chemistry levels there were appreciable gains, but the treatment time was protracted. A comprehensive physical examination of every patient including also pertinent laboratory tests should be done. Lobel believes that from results obtained by him and other workers further trials with vitamin A alone or combined with other vitamins seems definitely warranted. Detailed histories and audiograms of six cases are presented in the article.

ACOUSTIC TRAUMA

The final report on temporary deafness following exposure to loud tones and noise has been issued by Davis and colleagues,[57] of the Department of Physiology, Harvard Medical School.

Human ears were exposed to intense tones of frequencies 500, 1000, 2000 and 4000 cps at intensities of 110, 120 and 130 db. for periods of 1 to 64 minutes. After each exposure tests were made on the threshold of auditory sensitivity, and several studies were made of the distortion of pitch perception (diplacusis). Temporary impairment of hearing was regularly produced, but there was no evidence of cumulative injurious defects. The greatest defect occurs at half an octave above the exposure tone. With long exposures the hearing loss may be extensive for all tones above the exposure frequency. The monograph goes into detail concerning the average loss throughout the two octaves above the exposure tone. Recovery of hearing after exposure begins rapidly and then progresses more and more slowly. Recovery from a 60 db. loss may require four or five days to be complete. Recovery tends to be slowest for frequencies about 4000 cycles. Some men are much more susceptible than others to the production of hearing loss. The impairment of hearing produced by loud tones or noise is of the variable, or nerve deafness, type. The audiogram alone is not an adequate measure of the impairment of the auditory function. Exposure to a band spectrum (airplane noise) which causes a widespread hearing loss that is usually severer at the high frequency range is relatively ineffective in producing diplacusis.

Rüedi and Furrer [58] studied the effects of jet engines on the acuity of hearing. They noticed that airport personnel of jet propelled planes, "vampires," were affected with hearing disorders after a comparatively short time. These occurred in spite of the use of ear defenders occluding the external meatus. Measurements were made of the engines while on the ground both at idling and at full speed. The speed of the idling gas turbine was 3,000 rpm. The spectrum revealed a strongly stressed component of 850 cps which surpassed the general noise spectrum by 30 db. The oscillogram showed a nearly pure sinus wave of 850 cps. This noise is produced by the siren effect of the compressor, which has 17 blades and at 3,000 rpm yields 850 cps. The radiation of this tone is very directional, with special stress on the forward direction. At full speed 10,700 rpm, the siren tone is still present but is considerably lower in proportion to the remaining noise level, surpassing it by only 9 db. Four normal persons were subjected to this idling noise, two with, and the other two without, ear defenders for 10 minutes. The two without ear protection

57. Davis, H., and others: Final Report on Temporary Deafness Following Exposure to Loud Tones and Noise, Acta Oto-laryng., supp. 88, p. 1, 1950.

58. Rüedi, L., and Furrer, W: Special Kind of Acoustic Trauma by Jet Engines, A. M. A. Arch. Otolaryng. **54**:534, 1951.

showed a substantial hearing loss between 500 and 8000 cps, attaining its maximum at 1500 cps, a loss of 35 db., with the curves gradually rising again and returning to normal at 8000 cps. This effect is similar to the trauma produced by a pure tone at 800 cps with the exception of the C-5 dip, which characterizes the noise and report traumas. The history and findings of two mechanics subjected to this "vampire" jet engine noise for three and four years respectively showed the same changes, with losses starting at 500 and 1000 cps and reaching their maximum at 6000 and 12000 cps respectively. In both cases the C-5 dip is absent. The writers stress the point that ground and airport personnel who are not protected from this noise are in danger of acoustic trauma when subjected to idling jet engines. As a preventive measure they suggest the wearing of a close-fitting hard rubber cap covering both ears, which is more effective than an instrument introduced into the meatus. Speech becomes impossible in this noise environment; hence measures for the selection of certain frequencies important for speech are not necessary in this particular case.

Otological investigations of the effect of antiaircraft fire and protective measures are investigated by Schneider.[59] Cotton is unsatisfactory, but suitable defenders reduce trauma to 2%. Of the three Swiss models, the KTA defender fitted badly; "selectone" was suitable but should be fitted better. The Rüedi-Furrer model proved suitable for field service.

Prognosis of acoustic trauma due to gunshot was studied in a follow-up of 76 cases by Hamberger and Lindén.[60] After five years 6 of 76 patients had normal audiograms; 4 of these had recovered within three months. The authors conclude that no improvement may be anticipated after the first few months. Rupture of the membrana tympani seems to lessen the hearing damage.

Wheeler [61] made an attempt to overcome some of the physical and physiological variables in noise-induced hearing loss with a study of 35 men whose hearing was within normal limits and who were subjected to the same noise levels for the same length of time under similar environmental conditions. The purpose of this study as well as others which are being made now in other locations is to establish a systematic and quantified body of knowledge about noise-induced hearing loss. Our present knowledge of noise-induced hearing loss does not adequately explain two factors: (1) the incidence of loss in a known or sample of a population when the exposure variables are known, and (2) the rate of progress or the rapidity of the increase in loss after it has been detected in the person under known exposure conditions. To collect such information, certain requirements must be met for the exposure and the subject variables. The information regarding the exposure variables should consist of a known mean level of the noise during the time of study, the frequency characteristic of the noise, and the length of time the subject is exposed. The subjects should be persons with normal hearing acuity, preferably from the young adult age level, and incipient ear disease, predisposition to hearing impairment, and frequent exposure to high-level noises should be ruled out by a carefully taken history and examination. The occupation or trade of the subjects

59. Schneider, R.: Otological Investigations and Experiments with Ear-Defenders on Anti-Aircraft Personnel, Vrtljschr. schweiz. Sanitätsoff **27**:165, 1950.

60. Hamberger, C. A., and Lidén, G.: Prognosis in Hearing Injuries Following Acoustic Shot Trauma, Acta oto-laryng. **39**:160, 1951.

61. Wheeler, D.: Physical and Physiological Variables in Noise-Induced Hearing Loss, A. M. A. Arch. Otolaryng. **54**:267, 1951.

should be one in which the labor turnover is low. A group of men approaching these requirements was chosen from the apprentice groups at Mare Island Naval Shipyard. Thirty-five of 78 men originally chosen met the full requirements as to normal hearing. After a month interval 27 of the group of 35 were available for reexamination. This recheck showed a slight improvement in group hearing acuity at all frequencies except 4000 and 8000 cps. However, there were some variables, namely, better-fitting receivers in recheck, reduced room noise level in recheck and fewer seasonal ailments. This group also cannot be considered a random sample with respect to noise, as they apparently withstood successfully considerable exposure to noise as met with in the service and other conditions. This study, however, is one of a number, and it is hoped as more information is accumulated that it will be possible to evaluate the effect of potential bias.

In a study by Habermann [62] 200 telephone operators underwent otological examination by conversation, whisper, and Edelmann tuning forks; 16.5% showed retraction of the drum membrane of the operating ear, 6% of the other ear, and 12% of both ears. It was considered that the retraction in the operating ear was occupational. It caused only a slight elevation of the auditory threshold and loss of duration for the C-5 fork. The functional impairment was attributed to pathological consensual contraction of the middle ear muscles caused by continuous auditory stress due to occupation.

Fox [63] stresses the importance of acoustic trauma in industry and the indifference shown by many industrial leaders and insurance carriers to this important condition. Several suggestions have been advocated for the prevention of industrial or noise deafness: the use of ear protectors by the employees, the rotation of working personnel, and the planning of machinery and tool redesign to eliminate noise at its source. The last point seems the most desirable and may eventually prove to be the key to the solution of this problem. The writer feels, as many others do, that a realistic and scientific approach to this problem is indicated.

REHABILITATION

Statistics on the prevalence of deafness in England, Scotland, and Wales were gathered by Wilkins.[64] Sampling was done in a manner similar to that of Dr. Beazley of the United States Public Health Service. Thirty-two thousand persons were interviewed; 1,576 were found deaf, leading to the suggestion that there are 1,750,000 deaf in England, Scotland, and Wales. In addition, there are 80,000 persons with one defective ear, making a total of over 2,250,000 with some hearing defect. Only one-half have sought medical advice. This might well apply to other ailments.

The possibilities of auditory training of deaf and hard of hearing children is considered by Wedenberg.[65] He reports the results of a Swedish study, the work being done from 1946 to 1949 on 17 hard of hearing patients, training the hearing

62. Habermann, G.: The Influence of Telephoning on the Auditory Organ, Arch. Ohren-Nasen- u. Kehlkopfh. **157**:41, 1950.

63. Fox, M. S.: Acoustic Trauma, Wisconsin M. J. **50**:563, 1951.

64. Wilkins, L. T.: Prevalence of Deafness in England, Scotland and Wales, Acta oto-laryng. supp. 90, p. 97, 1950.

65. Wedenberg, Eric: Possibilities of Auditory Training of Deaf and Hard of Hearing Children, Acta oto-laryng. supp. 94, p. 1, 1950.

through the ear. A control group of 16 children with corresponding auditory impairment but educated in the regular school for the deaf was used. The results indicate that with the methods used it would have been possible to educate many of the pupils now in schools for the deaf to speak spontaneously, even if defectively, before reaching school age.

Whetnall [66] reports the experience of the clinic with which he is associated with the Medresco aid in running a deafness clinic. Details of the staff organization and procedure of the clinic are given. Emphasis is placed on the necessity for a team of workers, including otologists, teachers of the deaf, psychologists, speech therapists, and audiology technicians. Individually moulded inserts are fitted; auditory training and lip reading instruction are given, and printed instructions regarding the use of the aid are furnished. With the Medresco aid deaf children can be fitted to an extent not previously possible. The individual aid is of greater use than the group aid. With a loss greater than 60 db. children can acquire speech through hearing. Proposals are given for a training schedule for audiology technicians. Tables show the type and cause of the deafness, the ages and nature of the disability in the first 500 cases, also the results of follow-up inquiries. In spite of some obvious defects, a high opinion was formed of the Medresco aid after a year's clinical experience; 4,222 patients were fitted therewith.

Radley [67] describes the various elements influencing the articulation efficiency of an electrical system power distribution over the spectrum type of hearing loss and amplification characteristic. The experimental work in the Post Office Research Station is described. The conclusion is that in the majority of cases good results will be obtained with an amplification characteristic rising at 12 db. per octave up to 750 cps and flat or slightly tilting up for the higher frequency ranges up to 4000 cps. The amplification should amount to 40 db. The data on the Medresco aids are given.

([Abstracter's Note.] This same conclusion was arrived at by Hallowell Davis several years ago as the result of his work in Harvard Laboratory.)

A manual for the planning of a clinic for the rehabilitation of the acoustically handicapped, by M. Bergman [68] is published as *Supplement 89, Acta otolaryngologica.*

Stevenson [69] gives a year's experience with the government's hearing aid. The demand is greater than the supply; 7,100 were issued from July, 1948, to June, 1949. Government aid batteries are bulky but good and will last six weeks with 15 hours' daily use. Individual impressions for inserts are made. It is hoped a bone-conduction aid will eventually be available. The staff consists of five audiometricians, two mechanics, and an office staff. A training scheme is in action. Each patient is examined by an otologist. A medical psychologist, a neuropsychiatrist, a trained teacher of the deaf, and a hospital speech therapist assist.

66. Whetnall, E.: The Medresco Aid: Running a Deafness Clinic, Acta oto-laryng. supp. 90, p. 73, 1950.

67. Radley, W. C.: The Theory and Engineering Production of the "Medresco" Hearing Aid, Acta oto-laryng. supp. 90, 54, 1950.

68. Bergman, M.: Manual for Planning a Clinic for the Rehabilitation of the Acoustically Handicapped, Acta oto-laryng. supp. 89, p. 13, 1950.

69. Stevenson, R. S.: One Year's Experience with the Government Hearing Aid, Acta oto-laryng. supp. 90, p. 85, 1950.

A pure-tone hearing test, phonographically recorded, was given to 3,666 people in San Diego County [70] in the summer of 1948. Absolute thresholds were determined at five frequencies and masked thresholds at two. Auditory sensitivity was found to decline with age; women were more sensitive than men for the higher frequencies. Musically trained men and women had greater sensitivity than those without it. Twenty- to twenty-nine-year-old men averaged greater loss for high tones than those of the same age group tested before the war.

Adin and Shambaugh [71] report on long-term results in a series of 390 patients on whom fenestration operations had been done and who had been observed over a period of 5 to 10 years. Seventy per cent of these received and maintained a significant hearing improvement. Twenty per cent of the remaining 30% lost some of their improvement but still have usable hearing. Five per cent had no improvement at all, and 5% were considered to have bony closures. Ten per cent of the patients showed inner ear symptoms suggestive of labyrinthine hydrops. Postoperative serous labyrinthitis is now the principal cause of complete failure in fenestration operations at Northwestern University; 22 patients in this series had this complication. In 12 of these, bleeding was bothersome or more than average at the time of operation, an observation which confirms a view previously expressed by these writers that excessive bleeding is very important as a cause of postoperative serous labyrinthitis. Pregnancy following the fenestration operation does not seem to result in further cochlear nerve damage. Progressive cochlear nerve degeneration was noticed considerably more frequently in ears not operated on than in the ones operated on. The mechanisms of this partial protection provided by the fenestration operation is not known.

Thurlow and associates [72] report their third statistical study of auditory tests in relation to the fenestration operation. Special tests and pure-tone tests were administered to over 100 nearly consecutive patients before and after the fenestration operation. The results agree very closely with those previously reported. The spondaic word tests are highly reliable as tests for the threshold for speech. Auditory tests No. 9, descending levels, and No. 14, constant level, and the administration of "live voice" are practically interchangeable. The maximum articulation score is significantly higher when the lists are given by "live voice." However, articulation scores can be compared with one another only when determined by the same voice. The recorded tests for standardized testing for this reason are more advantageous. The maximum articulation scores showed no evidence of a hearing factor from one session to the next under the routine clinical methods of testing used. The scores were slightly lower for the patients with some high-tone nerve loss. The articulation score at the arbitrary level of loud speech is a less stable measure than the maximum articulation score. The size of the fenestra measured at operation bears no significant correlation to either the threshold for speech or the gain for speech following the operation. As in the previous studies, the average gain following fenestration is less for frequencies 2048 and 2896 cps than for frequencies 512

70. Webster, J. C.; Himes, H. W., and Lichtenstein, M.: San Diego County Fair Hearing Survey, J. Acoust. Soc. Amer. Lancaster **22**:473, 1950.

71. Adin, L. E., Jr., and Shambaugh, G. E., Jr.: A Study of Long Term Hearing Results in Fenestration Surgery, A. M. A. Arch. Otolaryng. **53**:243, 1951.

72. Thurlow, W. R.; Davis, H.; Walsh, T. E., and Eldert, E.: A Third Statistical Study of Auditory Tests in Relation to the Fenestration Operation, Laryngoscope **61**:124, 1951.

and 1024 cps. The gains at the lower frequencies agree very closely with the gain measured by auditory test No. 9. The relative deficit at the higher frequencies is still present after one year and is probably permanent. The maximum articulation score of either ear is not altered by the fenestration operation.

Pohlman [73] describes a new artificial ear drum which is more practical but somewhat less efficient than the diaphragm rod prosthesis reported by him several years ago.[74] This new artificial ear drum is a soft, conical tube made from a rubber-like plastic known as "Korogel." It comes in various sizes and shapes and can now be purchased commercially. The acoustic probe is first used to determine whether contact with the yielding portion of the middle ear will improve the hearing. The insert is then introduced, making contact with the proper area. The patient must be instructed in the technic of introducing and removing this prosthesis. A chronic discharging ear is not a contraindication as long as the patient realizes that this plastic drum must be removed every night before he retires and is also aware of the possibility of exacerbating symptoms which may arise and require medical attention. The artificial ear drum is an efficient substitute for the destroyed diseased middle ear apparatus. However, many patients aided by the acoustic probe cannot accept the plastic drug, since the sensitive area of the middle ear is partially concealed by an overhanging remnant of drum membrane preventing direct contact. Some surgical procedure should now be devised to deliberately leave a satisfactory middle ear and mastoid cavity that will accept some type of diaphragm or prosthesis.

Shambaugh [75] discusses three important steps in the management of any case of impaired hearing. These are a clear-cut accurate and complete diagnosis, correction of the hearing defect when possible, and audiologic rehabilitation when other therapy cannot restore practical hearing. He mentions the importance of the Rinne and Schwabach tuning-fork tests as the most important measures for differentiating a pure inner ear loss from a conductive loss with or without a superimposed perceptive component. The air-bone gap has also become an additional factor in making this differentiation. In a pure perceptive loss the air and bone audiograms interweave with no significant air-bone gap. However, in a lesion of the sound-conduction system there is a consistent air-bone gap of 20 db. or more. The size of the gap measures the degree of obstruction to sound condition, while the loss by bone measures the perceptive component. Shambaugh mentions the six main causes for conduction deafness: occlusion of the external auditory meatus; perforation of the tympanic membrane; Eustachian tube occlusion, including secretory otitis; suppurative otitis media; adhesive otitis media, and otosclerosis. A review of 1,046 cases of hearing impairment examined by Shambaugh and his associates in the year 1950 showed that 40% of the patients had pure perceptive deafness and 60% had a conductive loss. The largest group of cases in the conductive-loss class were 453 cases of otosclerosis; the second largest group were 53 cases of secretory otitis. There were 50 cases of adhesive otitis, 34 cases of chronic suppurative otitis media, and 27 cases of hearing loss due to perforation of the tympanic membrane. Two hundred five patients with otosclerosis chose surgery, and 53.4% were fully rehabilitated. In 40% of the entire group of 1,046 cases, substantial improvement could be attained by medical therapy. In view of this, Shambaugh feels that modern

73. Pohlman, M. E.: The Artificial Ear Drum, Ann. Otol. Rhin. & Laryng. **60**:117, 1951.
74. Pohlman, M. E.: The Artificial Middle Ear, Ann. Otol. Rhin. & Laryng. **56**:647, 1947.
75. Shambaugh, G. E., Jr.: The Management of Deafness, Illinois M. J. **100**:335, 1951.

otology has more to offer the hard of hearing patient than is sometimes appreciated. Inaccurate diagnosis is an important cause for failure to achieve maximum results from medical management.

THEORY

Payne and Githler [76] report their experimental findings of the effects of perforation of the tympanic membrane on cochlear potentials. The cochlear potentials were taken from cats that had portions of their tympanic membrane removed anteriorly, posteriorly, or in the region of the umbo. These areas were enlarged gradually and readings made until the entire membrane was removed. The results are reported as percentage losses of response for percentage of membrane removed. The results showed that regardless of where the perforation was located a loss occurs for every frequency. Small lesions in the posterior region of the membrane tested with frequencies of 100 to 2000 cps cause the greatest response loss; small lesions in the anterior portion, the next greatest loss, and small lesions in the umbo region, the least loss. Responses for frequencies of 5000 to 7000 cps were equivocal and very unstable. For the frequency of 10,000 cps the small anterior lesions produced the greatest loss, the posterior lesions the next greatest and the umbo lesions the least loss. The position of the small lesions is the chief determining factor of the response loss. For a given amount of membrane removed losses for frequencies above 2000 cps are less than for frequencies below 2000 cps until almost the entire membrane is gone. An increase in the size of the lesion does not alter the general shape of the response function until the entire membrane is removed.

The acoustical behavior of the head and the pinna was investigated by Onchi.[77] The ratio of reflected to diffracted sound on the head increases with higher frequencies. The sound pressure increment due to reflexion on the head increases at about 300 cps and attains its constant maximal value above 2000 cps. The acoustical behavior of the pinna in a sound field has not yet been measured accurately, and requires further investigation.

It is concluded that increased sound pressure on the head is one of the factors which brings about the discrepancy between the value of minimal audible threshold obtained by Wien's method (a sound source detached from the ear) and that by American methods (with a receiver on the head).

Bone-conducted sound waves set the basilar membrane into vibration when the membrane of the fenestrum ovale is less movable than that of the round window, according to Kraus.[78] All factors increasing the difference of mobility between these two membranes increase bone conduction. As the weights on a tuning fork increase the excursions of its limbs, the normal conduction operation of the middle ear increases the excursion of the stapes footplate, diminishing bone conduction. The external auditory canal and auricle act as a heavy resonator which damps the mobility of the conduction apparatus, by this means increasing bone conduction. Kraus thinks that this theory should explain all phenomena of transmission in the ear.

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76. Payne, M. C., Jr., and Githler, F. J.: Effects of Perforation of the Tympanic Membrane on Cochlear Potentials, A. M. A. Arch. Otolaryng. **54**:666, 1951

77. Onchi, Y.: Acoustical Behaviour of the Head and the Pinna, Otolaryngology, Japan **22**:14, 1950.

78. Kraus, M.: The Problem of Bone Conduction, Acta oto-laryng. **38**:233, 1950.

We are confronted with another theory of the recognition of sound direction by Kraus.[79] The author is of the opinion that the semicircular apparatus has a direct hearing- and sound-localization function. According to their direction, the sound waves excite different parts of the semicircular canals and their ampullae and thereby an impression of sound direction is created. The sound wave most efficient in provoking endolymph movements is one striking the semicircular canals tangentially. This theory is illustrated and "proved" by a simple experiment on a model. By comparative anatomy (fish have no cochlea but are able to recognize the direction of sounds) and by clinical observation it is known that strong acoustic stimuli can induce disturbances of equilibrium even in normal people and that people with unilateral deafness differentiate well between sounds from various directions. The intensity of sound is presumed to be perceived by the semicircular canals, while the cochlea analyzes sound and the brain coordinates all these several perceptions. The author feels that the old conception of the semicircular canals as an organ of "space acoustics" must be reconsidered. Other functions, such as equilibrium, perception and reflexes, are less important.

"Spinal pumping" (drainage) for congenital deafness is reported upon by Shugyo,[80] of Osaka, Japan. The effect of spinal drainage on the inner ear was studied in mammals. When there was bleeding into the acoustic nerve canal and cochlear aqueduct, it occurred also in the perilymphatic duct but not in the endolymphatic duct. The bleeding was believed to originate in the subarachnoid space. In most cases Reissner's membrane was relaxed. Corti's membrane was in contact with the organ of Corti in the lowest turns and in the lower half of the intermediate turns. After three or more cycles of pumping the hair cells atrophied and finally disappeared. The atrophy was most marked at the beginning of the second coil of the cochlea and diminished proportionately in both directions therefrom. This phenomenon suggests mechanical irritation exerted by the pumping affecting especially that portion of the cochlea.

Electrophysical studies on pigeons showed that the round window [81] transmits the sound to the cochlea, and the newly opened window permits only the compensatory liquid motion. The columella, the only middle ear bone in birds, had been fixed against the region of the oval window, simulating stapes fixation in humans.

Two tones of different frequencies were alternated successively five times per second.[82] A large difference gave the impression of two unrelated interrupted tones. A small difference sounded like pitch modulation of one tone. The transition point is called the trill threshold. For 14 subjects it was found to be a function of frequency but does not follow the pitch differential threshold. No practical application was suggested in the article.

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79. Kraus, M.: The Recognizing of Sound Direction, Arch. Ohren- Nasen- u. Kehlkopfh. **157**:301, 1950.

80. Shugyo, T.: Spinal Pumping for Congenital Deafness, J. Oto. Rhin. Laryng. Soc. **53**:159, 1950.

81. Van Eyck, M.: Function of the Round Window in the Transmission of the Sound Waves After Fenestration of the Labyrinth, J. Laryng. & Otol. **65**:183, 1951.

82. Miller, G. A., and Heise, G. A.: The Trill Threshold, J. Acoust. Soc. Amer. **22**:637, 1950.

Among 9,686 school children 7 to 14 years of age, the incidence of tinnitus was 0.587% and grew gradually higher as age advanced.[83] Only 10 per cent complained. The tympanic membranes were normal in 70 per cent. Hearing loss was found in 10%. It was not ascertained whether the tinnitus had any connection with the adenoids or other nose and throat disease.

The mechanism for auditory perception in vertebrates and invertebrates, especially insects, is discussed.[84] The various types of receptor apparatus for sound, their functional mechanism, and the mechanism for transmission are outlined. [This abstract is included for the benefit of those who are interested in comparative anatomy and physiology.]

Hood[85] presents studies in auditory fatigue and adaptation, with 40 graphs and two illustrations. This is a study in acoustics which hardly lends itself to abstracting but is mentioned here for those who wish to study it.

An attempt at solution of problems in acoustic physics is made in an article by Hood.[86] It does not lend itself to abstracting for clinical purposes.

Two papers[87] were not abstracted.

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83. Matsumoto, N., and Yamanobe, M.: On the Relation of Tinnitus to Age, J. Oto-rhino-laryng. Soc. **53**:10, 1950.

84. Von der Becke, A.: Auditory Perception in Man and Animals, J. M. Buenos Aires **3**:407, 1949.

85. Hood, J. D.: Studies in Auditory Fatigue and Adaptation, Acta oto-laryng. supp. 92, p. 8, 1951.

86. Hood, J. D ; Auditory Adaptation and its Relationship to Clinical Tests of Auditory Function, Proc. Roy. Soc. Med. **43**:1129, 1950.

87. Hood, J. D.: Studies in Auditory Fatigue and Adaptation, Acta oto-laryng. supp. 92. Transactions of the Second International Conference on Audiology, London, Acta. oto-laryng. supp. 90.

Abstracts from Current Literature

Ear

SECRETORY EFFUSION OF THE TYMPANUM. G. EDWARD TREMBLE, Laryngoscope **61**:791 (Aug.) 1951.

Secretory otitis media was first described by Politzer about 80 years ago. This condition is more prevalent today than previously. The reasons for this increased incidence may be (1) the widespread use of chemotherapy, before which the cases went on to suppurative otitis media, (2) the apparently more widespread incidence of virus infection, and (3) the increase in the popularity of plane travel.

If the ear is not treated properly, the fluid may remain behind the ear drum membrane for weeks or months. The condition occurs in almost any area and at almost any season, although it is more prevalent when the incidence of infections of the upper respiratory tract is higher. Anything that obstructs the Eustachian tubes may cause this condition. There may be infection or allergy. Malocclusion and nasopharyngeal growths must also be kept in mind. The patient complains of a blocked sensation and loss of hearing, and of feeling as though his head were in a barrel. Sometimes there is a crackling sound in the ear. The appearance of the drum membrane varies according to the amount and color of the fluid present and the degree of reddening.

The treatment consists of treating the underlying condition or cause, plus inflation of the tube in order that one may blow out the fluid. In most cases such treatment is successful, but in some paracentesis must be performed. Occasionally the latter must be done several times. After paracentesis, the fluid should be removed by suction or by other means. It must be borne in mind, however, that conservative treatment should always be tried first, inasmuch as 75% of the ears will respond to this.

HITSCHLER, Philadelphia.

THE STRIA VASCULARIS: REVIEW AND OBSERVATIONS. N. E. NACHLAS and M. H. LURIE, Laryngoscope **61**:989 (Oct.) 1951.

The literature on the stria vascularis is reviewed, and original experimental work is reported.

In the latter, an attempt was made to correlate the changes in the stria vascularis with the appearance of the organ of Corti and the contour of the scala media.

In specimens with inherited changes, abnormalities in the organ of Corti were accompanied with changes in the stria, but changes in the stria were not necessarily accompanied with changes in the organ of Corti.

HITSCHLER, Philadelphia.

CAPILLARY AREAS OF THE COCHLEA IN THE GUINEA PIG. C. A. SMITH, Laryngoscope **61**:1073 (Nov.) 1951.

Most of the work done on the blood supply of the inner ear has been concerned with the larger vessels. There is not much exact knowledge of the capillaries. Work on capillaries might enable us to learn more about the origin and absorption of the endolymph and perilymph, Ménière's disease, tinnitus, and hearing disorders. Much detailed information is given, including the cytology of the vessel walls and the innervation of the vessels.

HITSCHLER, Philadelphia.

OTITIS EXTERNA IN PUERTO RICO: A CLINICAL AND BACTERIOLOGICAL STUDY OF 82 CASES. MIGUEL ALONSO, Laryngoscope **61**:1114 (Nov.) 1951.

Otitis externa occurs more frequently in the summer, even in tropical countries. Of the many possible predisposing factors, Alonso believes that cerumen is the chief one and that swimming and bathing are of secondary importance only.

Fungi formerly were considered the principal causative organisms, but more recently the Pseudomonas aeruginosa and the Streptococcus have come into more prominence. Staphylococcus, however, was found to be present oftenest, with Pseudomonas next. The fungi played a relatively minor role. The acute cases generally showed the Staphylococcus, and the chronic cases, the Pseudomonas.

Treatment in cases of the staphylococcic infections consisted mainly of careful cleansing and drying and the application of sulfonamide powder or a bland ointment if the skin was excoriated. Instillations of acetic acid (5% or less) were used in the chronic cases.

HITSCHLER, Philadelphia.

EDUCATION OF HARD OF HEARING CHILDREN. A. JELLINEK, Nerv. Child. **9**:77 (Jan.) 1951.

This is one of several articles appearing in this journal in a symposium of "Problems of Speech Comprehension." Most of the papers in this number are informative and worth reading.

The child with a severe hearing disorder may not be able to develop speech spontaneously. The importance of early determination of hearing is stressed. If there is any doubt after the first year, auditory training should be instituted. The earlier in life one can supply lacking acoustic sensations through amplification by increasing hearing, the better the chance for speech development.

The use of hearing aids, even in an infant as young as 18 months, is desirable, primarily so that he may hear his own voice and thus stimulate his development through comparison with others. The author recommends the teaching of lip reading and use of hearing aids before the age of 3 years and estimates that 40 to 50% of deaf children may then be able to attend normal schools.

Children with hearing loss of slighter degree are often erroneously classified as retarded. Articulation of these children suffers unless their perception of sound is improved, through various devices. After initial training, a conscious effort must be made not to rely too much on lip reading. In other words, the visual aspects of speech should be minimized, and the child should be treated as if his hearing were normal.

Besides his training in hearing speech, the child must learn the source of sounds, and hence the warning values in terms of his own safety. Most children enjoy and are proud of their hearing aids, but children often have to be gradually accustomed to their use.

WEINFELD, Highland Park, Ill. [A. M. A. AM. J. DIS. CHILD.].

HISTOPATHOLOGY OF LATENT OTOANTRITIS IN THE INFANT. P. GIRARDET and J. P. CHRISTEN, Helvet. pædiat. acta **6**:71 (Feb.) 1951.

The work is based upon 176 histological examinations of the petrous portion of the temporal bone of 93 newborn infants, infants, and small children who died of various diseases. The anatomical peculiarities of the middle ear and of its accessory cavities are explained by means of serial sections and roengenological study after injections with iodized oil U. S. P. (lipiodol®). The authors discuss the term latent otoantritis, which seems to define best the clinical picture and the alterations observed. They make the following conclusions:

1. Latent otoantritis is a typical illness of newborn and young infants. It is found particularly often, by section, in infants 1 to 4 months old and less often in infants older than 7 months. In the second age group acute otomastoiditis is much more frequent. In small children latent otoantritis is rarely found.

2. Generally there are no perceptible clinical symptoms. From the histopathological point of view it is difficult to determine the clinical importance of the illness; it is characterized by relatively small lesions. The authors never observed collections of pus or necrosis of bones.

3. Latent otoantritis is not necessarily connected with dystrophy, since it is nearly as frequent in sthenic and newborn infants as in weak children. It depends chiefly upon the age of the patient. The special pathology of the upper respiratory tract of the young infant explains this fact, and the authors show the analogy between the pathogenesis of latent otoantritis and that of immature pneumonias in the first months of life. The topography of the Eustachian tube, middle ear, and antrum, the hospital surroundings, and the horizontal position of the infant during his meals are additional pathogenetic factors.

4. Latent otoantritis is nearly as frequent among infants without digestive disturbances as in infants suffering from such troubles. On the other hand, the latter show a higher proportion of severe and average lesions. No lesions have been found, however, which were so serious that they call for antrotomy. From the Authors' Summary [A. M. A. Am. J. Dis. Child.].

Tinnitus. J. P. Secretan, Pract. oto-rhino-laryng. **12**:290, 1951.

Tinnitus is evaluated by Secretan and classified into various categories. The extrinsic type, such as a pulsation of an aneurysm and articular cracklings of muscular spasm, can readily be determined. This type does not belong to the field of otology. Tinnitus associated with psychotic states is eliminated from this discussion. The types due to alteration in transmission, such as the presence of cerumen, acute and subacute catarrh of the Eustachian tube, or pathologic conditions of the middle ear, are usually easily diagnosed and quickly respond to specific treatment. True tinnitus is more or less of intrinsic nature, very often resembles a neuritis of the peripheral sensory nerve, and may present similar phenomena, such as paresthesia, anesthesia, or hypesthesia. However, this is not a simple classification, since in the latter group there is often a tinnitus which may be due either to hydrops of the labyrinth or labyrinthitis, which can confuse the picture in a true intrinsic type of tinnitus.

The author, in following Fowler's recommendation of employing a loudness balance test, has presented a series of cases in which he attempted to mask the tinnitus with a pure-tone audiometer. He has shown that the loudness balance test presents a tinnitus curve which follows closely the curve of deafness. He has also employed both the stellate ganglion block and the ablation of the stellate ganglion in a number of cases. His results are extremely striking in that the tinnitus is either reduced or completely relieved. However, he was not able to show in any of his cases any actual improvement in hearing acuity. He believes that further work along this line should be conducted and that in cases that fall into the group of neuritis of the eighth nerve the stellate block is a valuable adjunct to treatment. Persky, Philadelphia.

Clinical and Surgical Aspects of Latent, or Unrecognized, Otitis in Infants. T. E. Rossi and G. Bellieni, Minerva pediat. **3**:224 (April) 1951.

Unrecognized nonsuppurative otitis may be the causative factor of severe toxic enteric disorders. The diagnosis is difficult. The pathology, clinical symptoms, diagnosis, and treatment are discussed. Higgins, Boston [A. M. A. Am. J. Dis. Child.].

Radium Irradiation in Effective Treatment in Infections of the Nose and Throat in Children. H. A. E. Van Dishoeck, Maandschr. kindergeneesk. **19**:241 (Sept.) 1951.

Radium irradiation is an effective treatment in infectious foci in the nasopharynx causing tubotympanal catarrh. The principal consideration is combating the infection, and not the removing of a supposed mechanical obstruction of the tubal orifice by adenoid tissue. Tonal dips or high-tone loss is not influenced by this therapy.

From the Author's Summary [A. M. A. Am. J. Dis. Child.].

Pharynx

Radiotherapy of Adenoids in Infants. R. Clement and R. Bourdon, Lattante **20**:577 (Oct.) 1949.

The technique of irradiation of enlarged and/or infected tonsils and adenoids by x-rays and by radium is discussed. In the past 23 years at the Bretonneau Hospital, in Paris, irradiation of tonsils and adenoids has given satisfactory results in 66% of the cases and has been of no benefit in 6%. In 27%, there was functional improvement, although the adenoid and tonsillar tissues remained hypertrophied. Higgins, Boston [A. M. A. Am. J. Dis. Child.].

INDICATIONS FOR TONSILLECTOMIES IN PEDIATRICS. A. P. VERTIZ, Acta pediat. españ. **9**:686 (June) 1951.

This excellent appraisal of the indications for tonsillectomy is based on a study of more than 6,000 children upon whom operations were performed at Childrens' Hospital, México D. F., as compared with children with similar complaints who were not subjected to operation.

The author lists 12 supposedly valid reasons for tonsillectomy and rejects most of them. These indications are (1) local infection, (2) chronic hypertrophy, (3) recurrent tonsillitis, (4) frequent rhinitis, (5) otitis media and deafness, (6) prevention of infection in Waldeyer's tonsillar ring, (7) rheumatic fever, (8) prevention of other arthritic conditions, (9) nephropathies, (10) allergic diseases, (11) prevention of other acute infections, and (12) social reasons, such as mouth breathing and bad breath.

Vertiz concludes that unless the tonsillar tissue is actually diseased the tonsils should be retained as the first line of defence in childhood. Most of the reasons formerly held to be valid for removing tonsils, in early childhood at least, are shown statistically to be inadequate.

ALLEN, San Francisco [A. M. A. AM. J. DIS. CHILD.].

INDICATIONS FOR TONSILLECTOMY IN CHILDREN. B. WISKOVSKY, Pediat. listy **6**:8 (No. 1) 1951.

The following circumstances are listed as indications for tonsillectomy in children: (1) frequent recurrence of follicular (not catarrhal) tonsillitis, if it is not brought about by a high exposure to infection (mouth breathing, hemolytic-Streptococcus carriers in the child's environment); (2) protracted severe tonsillitis with unsatisfactory convalescence or with relapse within a short time (two to three weeks); (3) protracted peritonsillar abscess; (4) cases lacking a history of tonsillitis, only if the tonsils are hypertrophic to such an extent that they cause substantial limitation of the pharyngeal passage, or in the presence of signs of chronic tonsillitis (frequent formation of spots on the tonsils, stagnation of these spots, foul-smelling contents in the tonsillar follicles, peritonsillar adhesions, tonsils of unequal size, conditions after tonsillotomy); (5) diseases resulting from septic foci, with a history of tonsillar disease or with signs of chronic tonsillitis, and (6) exceptionally, cases with a normal tonsillar history and normal findings, if other infectious foci have been excluded by a thorough investigation.

FROM THE AUTHOR'S SUMMARY [A. M. A. AM. J. DIS. CHILD.].

Larynx

CONGENITAL ATRESIA OF THE ESOPHAGUS TREATED BY A ONE-STAGE PRIMARY ESOPHAGOGASTROSTOMY EMPLOYING A RIGHT TRANSPLEURAL APPROACH. JOHN L. MADDEN, J. Thoracic Surg. **21**:460 (May) 1951.

Congenital atresia of the esophagus is usually associated with tracheoesophageal fistula (90% of cases). In the remaining 10% of cases, those without tracheoesophageal fistula, the distal esophageal segment is either hypoplastic or entirely absent, precluding the feasibility of an end-to-end reconstruction of the esophagus. Determination of the presence or absence of an associated tracheoesophageal fistula is important. The presence of air in the stomach and small intestine indicates the probable existence of a tracheoesophageal fistula. Bigger has advised the use of iodized oil intratracheally as a confirmatory test.

MEYER, Chicago [A. M. A. AM. J. DIS. CHILD.].

CONGENITAL TRACHEO-ESOPHAGEAL FISTULA WITHOUT ESOPHAGEAL ATRESIA. JAMES A. HELMSWORTH and CHARLES V. PRYLES, J. Pediat. **38**:610 (April) 1951.

A 6-month-old infant had been hospitalized four times because of recurrent pneumonitis and because of coughing and cyanosis during feedings. Roentgenograms of the esophagus (with the patient in the supine and lateral decubitus positions) and endoscopy of the esophagus and the trachea failed to reveal a suspected fistula. Nevertheless, a diagnosis of tracheoesophageal fistula without atresia was made. The indicative signs were recurrent pneumonitis, coughing related to feeding, and meteorism. A rapid increase in abdominal distention after feeding and

coughing is a helpful corroborative sign. At operation, the diagnosis was confirmed and the fistula closed. The boy recovered.

MACDONALD, Pittsburgh [A. M. A. AM. J. DIS. CHILD.].

ADVANCES IN TREATMENT OF CROUP IN CHILDREN. A. WINDORFER, Deutsche med. Wchnschr. **75**:923 (July 14) 1950.

Croup due to unspecific micro-organisms, such as those seen in influenza, measles, scarlet fever, and pertussis, as well as croup due to diphtheria, may be cured by early treatment with penicillin. In all cases diphtheria antiserum and penicillin are given as early as possible. All of the 68 patients in this study could be cured. By this method of treatment mortality can be reduced further.

AMBERG, Rochester, Minn. [A. M. A. AM. J. DIS. CHILD.].

Nose

A TREATMENT PROGRAM FOR CHRONIC CHILDHOOD SINOBRONCHITIS. WALTER C. PRICE and VERONICA B. BINNS, J. Pediat. **38**:597 (May) 1951.

A comparative study of treatment of 54 children with chronic sinobronchial infection is presented. Roentgenograms of the sinuses before, during, and after treatment were the basis for preliminary evaluation. The authors conclude that penicillin used parenterally and intranasally (aerosol), in conjunction with hydroxyamphetamine (paredrine®) penicillin nose drops, and oral administration of methapyrilene (syrup of histadyl®), gives fair to excellent improvement in 95% of cases. The nose drops should precede the penicillin aerosol to open the airways.

MACDONALD, Pittsburgh [A. M. A. AM. J. DIS. CHILD.].

SINOBRONCHITIS IN CHILDREN. SAMUEL GEORGE SCHENCK and MORTON SELIGOWITZ, Radiology **67**:240 (Feb.) 1952.

The authors define sinobronchitis as a subacute or chronic infectious process involving the mucosa of the paranasal sinuses with secondary implantation of the infection upon the mucous membrane of the bronchi through postnasal drainage, and subsequent production of hilar infiltration and focal pulmonary lymphadenopathy. In 168 patients with this entity treatment was with x-rays, with full recovery in 58%, marked improvement in 28%, moderate improvement in 11%, and slight improvement in 3%. Case reports with roentgenograms of the sinuses and the chest are presented, and details of the x-ray therapy are given. Treatments are given to the sinuses and chest over a four-week period. The authors believe that sinobronchitis is a distinct clinical entity, and that x-ray therapy is the most effective treatment.

HODGSON, Rochester, Minn. [A. M. A. AM. J. DIS. CHILD.].

ACUTE RHINITIS IN SCHOOL CHILDREN. S. GARCIA VICENTE, Acta pediat. españ. **9**:606 (May) 1951.

The author discusses the benefits of discovering the presence of acute rhinitis before the sinuses are affected. There have been especially good results in the treatment of atrophic rhinitis before it develops into ozena, which is difficult to treat effectively.

ALLEN, San Francisco [A. M. A. AM. J. DIS. CHILD.].

MAXILLARY SINUSITIS AND THE POSTSINUSITIC LUNG SYNDROME IN ROENTGENOLOGIC PRACTICE. H. H. WEBER, Schweiz. med. Wchnschr. **81**:207 (March 3) 1951.

In cases of chronic cough, particularly in children, thickening or shadowing of one or both maxillary sinuses was seen in about 25% of the cases.

AMBERG, Rochester, Minn. [A. M. A. AM. J. DIS. CHILD.].

Miscellaneous

EFFECT OF ACTH AND CORTISONE ON THE UPPER RESPIRATORY TRACT. JOHN E. BORDLEY, New York J. Med. **51**:2636 (Nov. 15) 1951.

Since July, 1949, the nose, throat, pharynx, larynx, and lower airways of each of more than 60 patients have been studied. The work comprises 500 complete examinations during and after corticotropin and cortisone therapy in the department of otolaryngology at the Johns Hopkins Hospital. Changes observed were a decrease in nasal discharge, recession in swelling of the nasal mucous membrane, improvement in color of the mucous membranes, recession of polypi in the nose and sinuses, and a change in color and general appearance of lymphoid tissue in the nasopharynx. Even pemphigus was improved during the taking of corticotropin. Ménière's disease appeared to improve temporarily, but both these agents seem to lower the resistance of the mucous membranes to local infections, "especially in the nasal accessory sinuses. Patients with any history of sinusitis should always have adequate antibiotic coverage while under such hormone treatment." However, it is stated that neither of these agents offers much hope for permanent improvement in the treatment of allergies. Temporary improvement has been seen and reported hitherto. "However, the importance of such therapy should not be underestimated because of its transient effect."

VOORHEES, New York.

BANTHINE THERAPY OF TRIGEMINAL NEURALGIA. MELVIN H. STICH, New York J. Med. **52**:604 (March 1) 1952.

A white man aged 70 had had trigeminal neuralgia for three years. The increasing frequency and severity of the attacks seemed to call for surgical intervention. At first the right intraorbital area was chiefly involved. Then the condition spread to the distribution of the entire trigeminal nerve. Neuralgia of the fifth cranial nerve was the common diagnosis of all who examined the patient. The entire gamut of sedatives and antibiotics had been run, without relief. The patient said that he had never been sick before in his life. The family history was noncontributory. Neurologic examinations showed a completely normal state. Methantheline (banthine®) was prescribed in 100 mg. doses every six hours. No other medicines were allowed. The patient stated that within 40 minutes after taking methantheline all pain suddenly disappeared. None of the previous "trigger movements" which brought on an attack were manifest. The author stated that the patient had been on methantheline therapy for 10 months and that a 100 mg. dose gave complete relief for six to seven hours. Stopping use of the drug led to recurrence of attacks. Therefore, fearing recurrences, the patient continued to take methantheline. This drug is a true anticholinergic agent, having an inhibitory effect on the action of acetylcholine in the ganglia of the autonomic nervous system. It has been used against vagotonia, peptic ulcer, and spastic colon. This report seems to be the first record of its use in trigeminal neuralgia, which is "a disease of the Gasserian ganglion." The author asks others to try out this seemingly helpful remedy in trigeminal neuralgia.

Abstractor's Comment: Nothing is said of complications or sequelae which might follow the prolonged use of methantheline. Patients with this affliction will submit to anything to gain relief; therefore untoward conditions, if any, would not weigh much with them. One should, in all such cases, remember the brilliant work of Sluder in anesthetizing the Gasserian ganglion.

VOORHEES, New York.

DIFFERENTIAL DIAGNOSIS OF SEROUS MENINGITIS IN CHILDREN (WITH CASE REPORTS). FRITZ KUSKE, Arch. Kinderh. **139**:113 (No. 3) 1950.

The author presents a brief outline of the history, literature, and present opinions on differential diagnosis. Six cases are reported demonstrating difficulties in differential diagnosis. Diagnosis on the basis of analysis of the fluid is often not conclusive, since various etiological types may show identical cells. The most important task of the clinician is to determine the presence of otogenous or tuberculous serous meningitis as soon as possible.

GOTTSTEIN, Chicago [A. M. A. AM. J. DIS. CHILD.].

Books

The Temporomandibular Joint. Edited by Bernard G. Sarnat, M.D., M.S., D.D.S., F.A.C.S., in collaboration with Allan G. Brodie, D.D.S., Ph.D.; Harry Sicher, M.D.; John R. Thompson, D.D.S., M.S.D., M.S.; Joseph P. Weinmann, M.D., and Arnold A. Zimmermann, Ph.D. Price, $4.75. Pp. 148, with 52 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1951.

This monograph, consisting of seven chapters, is the result of the coordinated efforts of the above group of anatomists, pathologists, and directors of several departments of dentistry and orthodontia. The first chapter describes structures comprising the temporomandibular joint, muscles of mastication, and the suprahyoid muscles involved in mandibular movement. It presents a clear and understandable description of these complicated processes. The second chapter deals with the histology and embryology of joint cartilages, condyle, and mandible. The third chapter lists the growth factors of all bones of the face and the temporomandibular joint. Infections, arthritis, and trauma are given as causes for ultimate deformities. Further bases for pathology of the joint appear in chapter 4, including vitamin deficiency, glandular disorders, and two types of arthritis.

The first half of chapter 5 is a description of all structures regional to the temporomandibular joint which may have been involved in the production of the symptom complex known as Costen's syndrome. The entire anatomic and functional basis offered by Costen is termed erroneous by the author, who considers Costen's illustrations "diagrammatic misrepresentations," and who concludes that "the syndrome as such should be abandoned." Only one of Costen's anatomic explanations is conceded to be correct by the author. It appears to this reviewer that a more careful reading of Costen's material would have resulted in a less destructive attitude toward his work.

Chapter 6 is devoted to the dynamic equilibrium among the muscles which control the mandible. The closing chapter details the diagnosis and dental management of five classic cases of malocclusion and related symptoms within the temporomandibular joints. It is sometimes difficult in reading the last chapter to distinguish between the symptoms listed in the authors' cases and those suggested by Costen.

CORRECTION

Correction is made of the error that appeared in the review, in the March issue of the A. M. A. Archives of Otolaryngology, of the book entitled "Audiométrie vocale," by J. E. Fournier, who is Director of the French Company of Audiology, and not Clinical Professor of Otology, Rhinology, and Laryngology, of the Medical Faculty of Paris. The latter title belongs to Prof. A. Aubin, who wrote the preface to the book.

Directory of Otolaryngological Societies *

INTERNATIONAL

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico, Time: January, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3, Pa.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: 1953.

Fifth International Congress of Oto-Rhino-Laryngology

Place: Amsterdam, Netherlands. Time: June 8-15, 1953.
President: Prof. Eelco Huizinga.
General Secretary: W. H. Struben, J. J. Viottastraat 1, Amsterdam-Z.
Scientific Secretaries: J. Bijtel and P. G. Gerlings.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 6-10, 1952.

American Broncho-Esophagological Association

President: Dr. Fred W. Dixon, 1027 Rose Bldg., Cleveland.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Roosevelt Hotel, New Orleans. Time: April 28-29, 1953.

American Laryngological Association

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 7.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Place: Waldorf-Astoria, New York.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Place: Academy of Medicine, Atlanta.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Place: Indianapolis Athletic Club, Indianapolis.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.
Place: County Medical Building, Los Angeles.

American Otological Society

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Louis Joel Feit, 50 Park Ave., New York 16.

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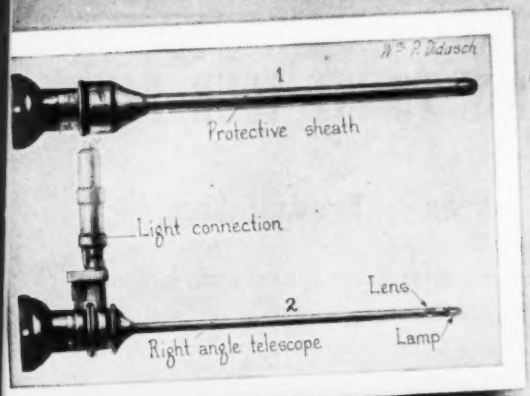

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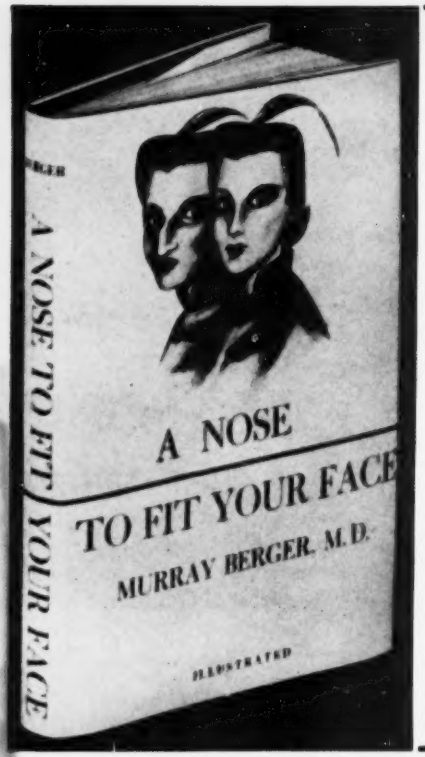

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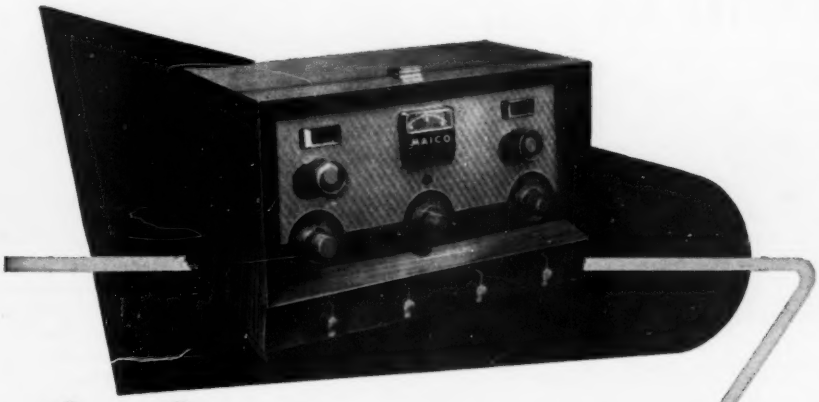

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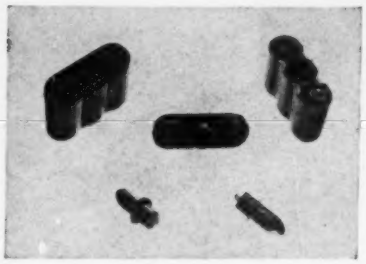

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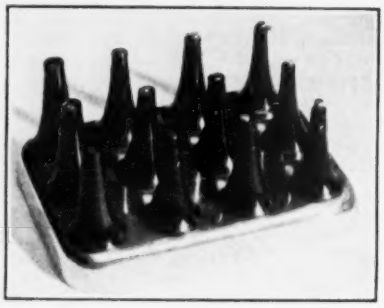

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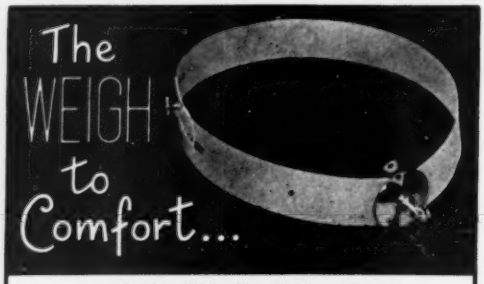

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The new DeVilbiss Respirator #10, priced at only $3.50 retail, can quite possibly prove of benefit to your patients who are sensitive to nuisance and allergenic types of dust.

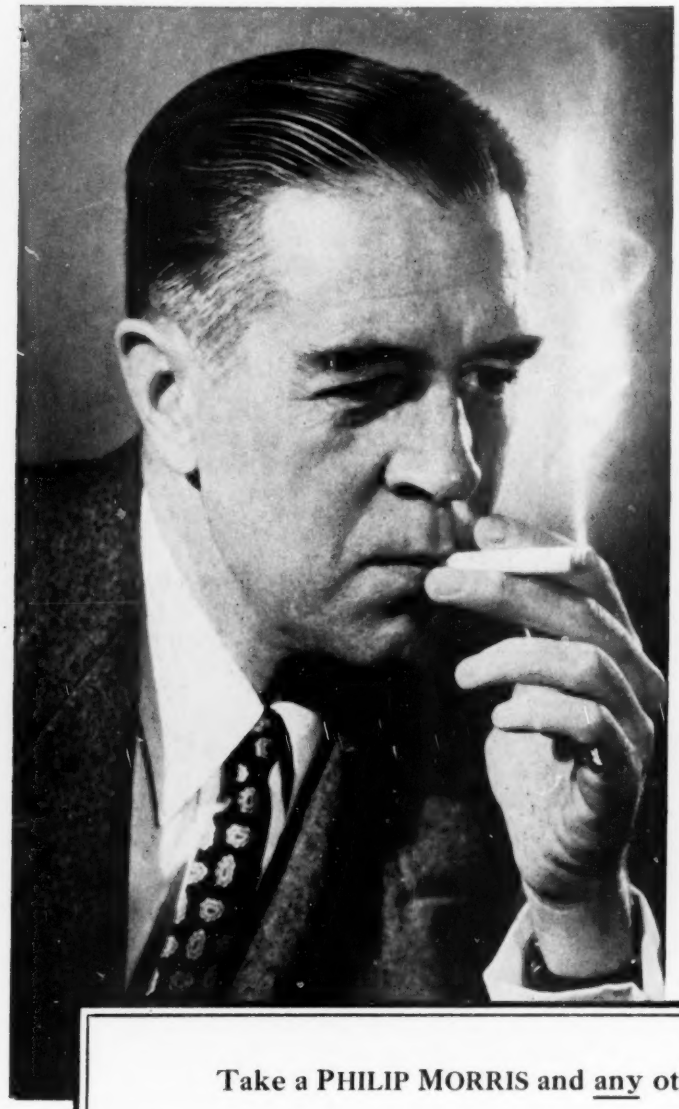
Tests show DeVilbiss Respirator #10 to be 99.2% efficient in filtering particles ranging from 15 to 60 microns.

Easy to clean and adjust to the face. Light, comfortable, and with a large breathing area. Now being stocked through all leading drug stores.

For further information, write to Respirator Department, DeVilbiss Company, Somerset, Pennsylvania, or Windsor, Ontario.

Tell your patients about the

DeVILBISS RESPIRATOR # 10



Doctor, be your own judge... try this simple test

With so many claims made in cigarette advertising, you, Doctor, no doubt prefer to judge for yourself. So won't you make this simple test?

Take a PHILIP MORRIS and <u>any</u> other cigarette

1. Light up either one first. Take a puff—get a good mouthful of smoke—and s-l-o-w-l-y let the smoke come *directly* through your nose.

2. Now, do exactly the same thing with the other cigarette.

You will notice a distinct difference between PHILIP MORRIS and <u>any</u> other leading brand.

PHILIP MORRIS

Philip Morris & Co. Ltd., Inc., 100 Park Avenue, New York 17, N. Y.

NEO-SYNEPHRINE®

HYDROCHLORIDE

. . . reduces nasal engorgement . . .

. . . promotes aeration . . . encourages drainage

A few drops of Neo-Synephrine 0.25% in each nostril will promptly check mucosal engorgement and hypersecretion, promoting greater breathing comfort over a period of several hours.

The resultant relief to the hay fever sufferer is decidedly gratifying. Prolonged action of Neo-Synephrine makes fewer applications necessary, consequently longer periods of rest and sleep are possible.

Neo-Synephrine does not lose its effectiveness on repeated application and may, therefore, be relied upon to give relief throughout the hay fever season.

Neo-Synephrine is practically free from sting and compensatory congestion; does not appreciably inhibit ciliary activity. Neo-Synephrine has been found relatively free from systemic side effects such as nervous excitation, cardiac reaction or insomnia even when tested on hypertensive, cardiac and hyperthyroid patients.[1]

Supplied in 0.25% solution (plain), bottles of 1 oz., 4 oz. and 16 oz.; 0.25% solution (aromatic), bottles of 1 oz. and 16 oz.; 0.5% solution, bottles of 1 oz.; 1% solution, bottles of 1 oz., 4 oz. and 16 oz.; 0.125 (⅛)% solution, bottles of ½ oz.; 0.5% water soluble jelly, in ⅝ oz. tubes.

1. Van Alyea, O. E., and Donnelly, Allen: *Arch. Otolaryng.*, 49:234, Feb., 1949.

Winthrop-Stearns INC.
NEW YORK 18, N. Y. WINDSOR, ONT.

Neo-Synephrine, trademark reg. U.S. & Canada, brand of phenylephrine.